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CONTENTS

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EDITORIAL

RELATIONSHIP OF BIOPSY TO DIAGNOSIS

JOHN A. WAGNER, M.D.[1]

It is now just 100 years since the idea of biopsy was introduced into the Western Hemisphere by Francis Donaldson of Baltimore. The notion that cancer could be diagnosed by the removal of pieces of tissue from the living subject had apparently been conceived in France and upon his return to America after a period of postgraduate study, Donaldson set down precise and lucid directions for the study of suspected malignant tissue, for the technique of aspiration biopsy and for the interpretation of cells scraped from the surface of a suspected cancerous lesion. The idea of biopsy, therefore, is not new.

In 1853, resistance to this innovation was apparently very great, for the cell theory of disease had not been crystallized, and indeed, the microscope was still considered by many physicians as a toy. During the ensuing century, biopsy has become an established technique in the management of neoplastic disease, largely through the development of the specialty of pathology, improvement in sectioning techniques such as the development of the microtome, and lastly, through the application of new staining techniques. The principle of the biopsy, however, has not changed. We have been able to produce better sections for study quicker, with better stains, and we are able to study these specimens more readily through improvements in the optical mechanisms of the microscope.

At the time the idea of biopsy was conceived, pathology as a specialty of medicine, had not yet evolved. With the development of departments of pathology in the medical school, application of this specialty to the management of disease gradually spread to hospitals not necessarily associated with schools of medicine. Thus, medicine gradually came to recognize the pathologist as the "doctor's doctor" and has progressively embraced the advice and counsel of these specialists in the management of neoplastic disease. Progress has been slow, and indeed, within only recent decades have the majority of the hospitals in the Western Hemisphere sought the advice and the services of a trained pathologist.

As momentous as these improvements may seem in the face of the discovery of Donaldson, the art of biopsy has not changed. We still employ the three methods originally described; namely, the removal of a small sample of the suspected growth, an aspiration of a portion of it, or the scraping away of cells from its surface. While each of the three

[1] Editorial Board, Maryland State Medical Journal.

techniques has certain advantages, an analysis of the accuracy of any of these methods, when applied to a single clinical problem, tends to eliminate aspiration biopsy and smear techniques as being too general. A diagnosis of any particular suspicious lesion rests first on the clinician's appraisal of it through a thorough physical examination and next upon his training in the gross recognition of a suspected malignant growth. Next, he must be inclined to proceed toward the removal of a portion of it for microscopic study. Next, his skill in sampling the suspected area is a most important adjunct to an accurate pathologic diagnosis. All pathologists will agree readily that the quality of a biopsy is an index to an accurate diagnosis and that the skill and understanding demonstrated by the surgeon performing the biopsy reflects directly into the quality and usefulness of the pathologic diagnosis.

Aspiration biopsy and cytologic techniques are not new. They are not to be condemned; indeed, there are certain instances where both have a definite application either through mass survey research problems or in instances where direct vision biopsy is not possible. The concept that these techniques are new is also to be refuted. True, they have been improved, but the improvement has largely been through staining techniques.

The physician who is faced with the problem of the diagnosis of a suspected malignancy must face clearly the necessity of a careful course through enthusiasm stimulated by recrudescent activity and interest in less accurate methods of study. He should not be influenced particularly by staining techniques, variations in the basic structure of the microscope, and perhaps other innovations. He must remember that none of these has radically changed methods of medical diagnosis.

One should not close his ear and eye to progress, for indeed, a method might appear which would improve upon basic biopsy technique which depends upon the inter-relationship and cooperation from the patient who must first be taught to detect an abnormality and report it to the doctor. The physician must be taught always to remember thoroughness in his physical examination, and next, he must remember the science and art of biopsy. Next, the services of a competent trained pathologist is absolutely necessary for an authoritative and complete study of the problem.

It is not therefore the techniques employed in biopsy which are important but rather a patient-surgeon-pathologist relationship based upon scientific training, care, thoroughness, and skill. There is no substitute for a good physical examination. There is no substitute for a well selected biopsy. There is no substitute for an authoritative opinion of a competent pathologist.

CONGRATULATIONS TO THE BALTIMORE COUNTY MEDICAL ASSOCIATION

The Baltimore County Medical Association has published a Directory containing the names of the members of the Baltimore County Medical Association, with their addresses, telephone numbers, and specialties. This has been set up under localities and alphabetically. It also contains a section called, "Information of Medical Interest." This gives the names of many of the druggists by specific localities, the names of the employees of the Baltimore County Health Department, the Maryland Bureau of Medical Services and Hospitals, and the Hippocratic Oath. This is a well compiled, attractive booklet, containing valuable information.

Scientific Papers

IRON DEFICIENCY ANEMIA[1]

PHILIP F. WAGLEY, M.D.[2]

These comments are presented in four sections. The first consists of remarks on iron metabolism. The second deals with the clinical and hematological features of iron deficiency anemia. In the third section a clinical entity is mentioned that may simulate simple iron deficiency anemia, and the fourth section is devoted to therapy.

Various estimates on the so-called average daily diet place the iron content of such a diet at 5 to 17 milligrams. Some of this iron is not readily available for absorption. Some is in organic form as in porphyrin compounds and much is present as colloidal ferric hydroxide. How much iron is absorbed depends upon a variety of factors, such as the amount of acid gastric secretion, phosphates and calcium in the diet, and the amount of ascorbic acid and other reductants in the food. The acid gastric juice serves two purposes. First, at the low pH of the gastric content the ferric hydroxide molecules which tend to aggregate and polymerize are dispersed. Subsequently the ferric ions are reduced to ferrous ions at a low pH and it is in this reduction that ascorbic acid and other reducing substances are important. The ferrous salts are more soluble than the ferric salts. If the ferric ions are not reduced and absorbed, they may precipitate out with phosphate. It is apparently in the ferrous form that most iron is absorbed through the intestinal mucosa (1). Some experiments show that as much as 15 times more ferrous iron is absorbed than in the ferric form (2).

There are other factors not clearly understood that apparently regulate iron absorption. Now iron absorption is being emphasized because the body has no way of losing large amounts of iron except by blood loss (3). It has been estimated that the body may lose about 1 milligram of iron per day due to traces of iron in the bile, and still smaller quantities in the urine; apparently some loss occurs by sloughing of mucosal cells in the gastro-intestinal tract. Once iron gains admission to the body, it cannot under normal circumstances be excreted in large amounts. Injection of iron intravenously is not followed by excretion of any increased quantity, either in the urine or in the feces. Therefore, if the loss of iron from the body is only about 1 milligram a day and iron in the diet is in a range roughly around 10 milligrams a day, then normally the per cent of iron absorption in an adult male should be in the range of 10 per cent.

Obviously, it is important to understand the factors that maintain the balance between absorption of iron and the body requirements for iron. Numerous studies have been carried out to elucidate this important problem. In experiments in dogs it has been shown with radioactive isotope technique that the feeding of iron to a normal animal is associated with very little absorption. However, if the same amount of iron is fed to a chronically anemic dog (made so by bleeding) the iron is absorbed in amounts 5 to 20 times that retained normally (4). This observation has been confirmed in humans (5). This seems like a straight forward and what we would consider a normal adjustment. However, it is more complicated than it would seem to be at first. If an animal is made acutely anemic, by blood loss, and is then fed iron by mouth, the

[1] Read before the one hundred and fifty-fifth annual meeting of the Medical and Chirurgical Faculty of the State of Maryland.

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iron absorption during the first few post-anemic days is not increased (6). This apparent discrepancy between the amount of iron absorbed by an animal that is chronically anemic in contrast to one that is acutely anemic has suggested to some workers that the amount stored in various tissues of the body may control iron absorption. Thus, if a large amount of iron has been stored and is still available for hemoglobin formation it was argued that very little iron would be absorbed (6). However, as those iron stores were depleted, more and more to make hemoglobin, as in the chronically anemic animal, then iron was absorbed more readily through the intestinal mucosa. Such teleological reasoning led to more detailed study of plasma iron which is assumed to be a transport form of iron serving as a relay from the storage depots in the reticuloendothelial system and in some parenchymal cells in the body to the bone marrow. Ordinarily plasma iron circulates in a bound form with a Beta I globulin. Normally this protein is present in concentrations of 250 to 300 milligrams per cent, one-third of which is bound with iron. The normal plasma iron level is in the range of 70 to 140 micrograms per cent. This iron level fluctuates in various types of anemia but is low in iron deficiency anemia (7). It was thus assumed that as the storage iron was depleted and used up in the formation of hemoglobin in animals bled chronically, the plasma iron began to fall and the absorption of iron was increased. This seemed to be a rational explanation until it was found that if one increases the level of plasma iron temporarily to saturation by injecting ferrous gluconate intravenously, just before feeding radioactive iron by mouth in a chronically anemic animal, there was no decrease in the rate of absorption of the radio iron (8). Furthermore, one can inject iron free Beta I globulin, that is the carrying protein for plasma iron, and thereby raise temporarily the iron binding capacity of plasma without, however, affecting greatly the actual absorption through the intestinal mucosa (1). It seems we have to assume that the plasma iron is simply a passive substance, the level of which does not influence appreciably the movement of iron from the intestines into the blood stream. Therefore, it is apparent that an essential aspect of iron absorption is still not understood.

Let us turn our attention now to what happens to iron after it is absorbed. It apparently enters the mucosa as a ferrous ion and is incorporated with a protein that has been named apoferritin to form an iron containing compound named ferritin. It is then carried by plasma in combination with Beta I globulin in the ferric form to the bone marrow or other depots in the body. The plasma iron level does not seem to vary with age or between the sexes or with normal menstruation. There are roughly about 4.5 grams of iron in the human body. Sixty to 70 per cent of this is incorporated in hemoglobin. Three to 5 per cent is in myoglobin. Less than 1 per cent is incorporated in certain enzymes such as cytochrome oxidase, peroxidase and catylase. About 15 per cent of the iron, or one-half of that unaccounted for in the above forms, is in the form of polymerized ferric hydroxide units contained in ferritin and in hemosiderin (5).

This brings us to the mention of what has been called storage iron. As stated previously, iron may be temporarily held in tissues in the form of ferritin or hemosiderin. To recapitulate ferritin is a combination of ferric hydroxide polymerized micelles with a colorless crystallizable protein called apoferritin. Hemosiderin is apparently larger aggregates of similar material which may be easily seen microscopically (1). It is assumed that such iron moieties are available to the organism when needed for hemoglobin formation. Estimates of normal stores have been calculated and apparently such storage depots of labile iron do not become ordinarily very large (9).

So much for storage iron and to launch on the topic of hemoglobin per se would be outside the scope of this paper, so let us list the potential causes of iron deficiency. Obviously, the first is

blood loss. The second is inadequate intake of iron. This may be seen in individuals on a restricted diet, either from poverty, regional dietary characteristics, or idiosyncrasies of the individual. There may be an increased demand for iron. For example in children as their blood volume increases, the amount of iron intake may be deficient and the child develop an iron deficiency anemia. Also in pregnancy several hundred milligrams of additional iron must be absorbed. Another cause for an iron deficiency anemia is impaired absorption of iron even when the intake may appear to be adequate such as in sprue, steatorrhea, and chronic diarrhea. From the remarks made previously it is apparent that achlorhydria would contribute to the impairment of iron absorption in any of these conditions.

Now in the characteristic clinical picture of iron deficiency anemia, the onset is insidious. Symptoms, on careful questioning, may date over a period of several years, prior to the establishment of the diagnosis. Hypochromic microcytic anemia is much more common in females than in males. The age range of highest incidence is between 30 and 50 years. As is common to all anemias, these patients have weakness and easy fatigability. Headache and insomnia are common complaints. Gastro-intestinal symptoms occur frequently. In approximately one fourth of these patients there is the complaint of glossitis and sometimes stomatitis. This soreness of the tongue and mouth is usually not intense, but occasionally is associated with some pain on swallowing.

As you know, physical examination of these patients usually reveals a rather listless individual in no acute discomfort. The skin and mucous membranes are pale, but there is no icterus. Such a patient may show very little weight loss and may actually be obese. There may be chylosis. Occasionally there is slight papillary atrophy of the tongue and even small ulcers on the buccal mucous membranes. There may or may not be very slight cardiac enlargement. Apical systolic murmurs are not uncommon. The spleen is palpable in about one-third of these cases. However, it does not become very large. In women occasionally one sees spooning of the nails or koilonychia.

The laboratory studies are diagnostic. As you know, the anemia of iron deficiency is characterized by a hypochromic microcytic red blood cell. The hemoglobin concentration in each red cell is low and the overall cell size is decreased. There may be a moderate amount of variation in size and shape of these red blood cells. Characteristically, this variation in size and shape does not become marked. Reticulocytes, punctate basophilia of the red blood cells and nucleated red blood cells are rarely seen unless there has been a recent acute blood loss. The white blood cells and platelets are unaffected. In the absence of iron it is obvious that hemoglobin cannot be synthesized and there is a maturation arrest at the normoblastic stage of erythropoiesis. The bone marrow, therefore, usually appears hyperplastic showing a predominance of normoblasts. Many of the nucleated red blood cells in the bone marrow contain small amounts of hemoglobin, but not enough to mature normally and consequently polychromatic normoblasts are found in increased number. There is no evidence that there is an increased rate of destruction of red blood cells in iron deficiency. The serum bilirubin and icterus index are either normal or actually diminished. The urobilinogen excretion is decreased. The life span of the red blood cells in simple iron deficiency anemia is normal. As to other laboratory studies, the urine is characteristically not remarkable. The stools should be examined repeatedly for the presence of occult blood. It should be emphasized at this point that not infrequently individuals who are losing blood through their gastro-intestinal tract may have intermittently guaiac negative stools. Consequently, repeated examinations of the stools for occult blood should be made in such individuals if the source of blood loss is not obvious.

Among the conditions that might be confused

occasionally with simple iron deficiency anemia one should be mentioned here. That is thalassemia. Although severe cases were first described in 1925 (10) and separated as a disease entity from other types of anemia, it has not been until more recently that less severe instances of this disease have received a good deal of attention. Various groups have described what on first glance was a simple iron deficiency anemia, but which on further study revealed a familial tendency and was refractory to iron therapy (11, 12). Now such cases can be differentiated from simple iron deficiency anemia on the following bases. First, these patients or their ancestors usually come from the Mediterranean area. Secondly, on close questioning, or on study of members of the family, it may be found that unsuspected anemia exists among some relatives. In the severe cases the patients frequently have prominent malar eminences and epicanthal folds giving them a Mongoloid appearance of the face. In addition to pallor of the mucous membrane there may be slight icterus. The spleen is enlarged and may be sufficiently so to extend into the lower quadrants or across the midline. Less frequently, the liver is enlarged. The blood examination is quite striking. In addition to the anemia, in the severe cases, the reticulocytes are increased usually over 4 per cent. The platelets are normal in number and appearance. Frequently the white blood cell count is increased moderately and there may be a shift to the left with myelocytes and an occasional myeloblast. The most impressive change, however, on blood smear is the basophilia, stippling, nucleated red blood cells, and the appearance of the mature red blood cells. There is marked variation in size and shape of the erythrocytes with extreme pallor. Occasionally there is an accumulation of hemoglobin within the center of each cell giving it the appearance of a target cell. Now many cases have what has been described in a very mild degree with the hemoglobin ranging around 10 grams per cent. In the severe younger group of cases no really effective treatment has been discovered. These patients do not respond to iron therapy. Occasionally they seem to improve with splenectomy, but usually in the markedly anemic individuals, the course is a progressive one and the patients die of some intercurrent infection.

And now as to therapy of simple iron deficiency anemia. First and foremost it is obvious that if the patient is losing blood the source of blood loss should be treated as adequately as possible. Secondly, any chronic infection that might be present should be cleared up. For, if a person has an iron deficiency anemia, the presence of a chronic infection will impair the utilization of any iron given to that patient. Thirdly, the specific therapeutic measure is the administration of iron and it should be emphasized first and foremost that this administration of iron can practically always be carried out by oral dosage. And now what forms of iron can be used? The most popular type of iron administration is, as you know, ferrous sulphate in 0.2 gram tablets. From 3 to 6 tablets should be given per day. The medicine should be administered immediately following each meal. It should not be given on an empty stomach as it is likely to be irritating to the gastric mucosa in such concentrations. It is advisable to start out with small dosages and then increase as the tolerance allows. Ferrous gluconate is less irritating to the stomach and can be given in the same amounts. When such preparations are employed, it is not necessary to give as large amounts as are used of ferric ammonium citrate or ferrous carbonate. To treat a person with ferrous sulphate or ferrous gluconate, only costs the patient around four cents a day.

At the present time, unfortunately, there is the tendency to give "shotgun" therapy in anemia. For example, the patient may be given small amounts of iron as well as folic acid and vitamin B12 with certain other vitamins by mouth. Such therapy is not only ineffective, it is actually dangerous. In addition it is quite expensive. Some such preparations cost the patient as much as forty cents a day. In the literature, recently, quite a bit of work has been reported on cobalt

(13). It has been suggested as a possible supplementary therapeutic measure. Suffice it to say that despite some of the reports as to its hemopoietic activity, cobalt should not be used in simple iron deficiency anemia. It is very likely to cause anorexia, an unpleasant metallic taste, nausea, and vomiting.

One point should be made in this discussion and that is in iron deficiency anemia iron and iron alone by mouth is the necessary therapeutic measure. The administration of iron parenterally is practically never indicated. Such a therapeutic injection is not necessary and it is likely to have unpleasant side effects. These toxic symptoms consist of flushing, palpitation, sometimes precordial pain, nausea and vomiting. The response to iron in an uncomplicated case of iron deficiency anemia is characteristic. After a week or so of treatment the reticulocytes begin to rise. Following the reticulocyte peak the hemoglobin level increases. The rate of increase is sometimes as high as 0.2 gram per cent of hemoglobin a day. As normal values are reached, the rate of hemoglobin increase diminishes. Unless the case is complicated by continuing blood loss, or by infection or by another deficiency, the blood should be close to normal range within six weeks.

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THE ACUTE ABDOMEN*

PHILIP THOREK, M.D.†

The subject of the acute abdomen will always present an interesting challenge to the practitioner and surgeon alike. I have examined charts from the surgical services at the Cook County Hospital for a period of fourteen years, the purpose being to determine which diseases are most frequently mistaken in the acute abdomen. To my surprise I did not find fifty or seventy-five conditions which confuse us, but rather six

* Courtesy of the Maryland Academy of General Practice. Presented before this Association at its Annual Scientific Assembly, October 9, 1952, Lord Baltimore Hotel, Baltimore.

† From the Departments of Surgery, University of Illinois, Cook County Graduate School of Medicine, Cook County Hospital, American Hospital and Alexian Brothers' Hospital, Chicago, Illinois.

outstanding ones that we mistake most frequently. These six conditions are:

1. Acute Appendicitis
2. Acute Cholecystitis
3. Perforated Peptic Ulcer
4. Acute Hemorrhagic Pancreatitis
5. Renal Colics
6. Coronary Disease

There is a seventh disease which deserves special consideration, namely, salpingitis. Acute or chronic salpingeal pathology is frequently associated with a perihepatitis which produces pain in the right upper quadrant (pseudo-gallbladder pain). Because of this, gallbladder explorations and other surgical procedures have been done in cases of salpingitis, resulting in danger to the patient and embarrassment to the surgeon.

To make a diagnosis one must have a simple and workable plan in mind. Our plan consists of four headings, namely, history, present symptom complex, physical examination and laboratory data. This routine has served us well and we utilize it daily.

Acute Appendicitis

The more one sees of acute appendicitis, the more one respects the condition. The statement "only an appendix" is indeed a dangerous one. This condition is more frequently found in individuals under the age of forty and is somewhat more common in males. It will be recalled that gallbladder conditions appear most frequently after the age of forty. The story the patient relates is usually quite stereotyped. To put it in his language: "Something I ate gave me a belly-ache." This is his way of describing acute epigastric distress. When he gets his "belly-ache" he often attempts to obtain relief with either a cathartic or an enema. Within the first twenty-four hours his "belly-ache" becomes a soreness low on the right side. His acute epigastric distress has become localized to the right lower quadrant. The "two-question test" is both useful and time-saving. Question Number 1: "Where was your pain when it started?" to this interrogation the patient points to his entire abdomen. Question Number 2: "Where does it hurt you now?"; he then points to the right lower quadrant, usually McBurney's point. This simple method of having the patient demonstrate diffuse pain which localizes to the right lower quadrant will diagnose the vast majority of cases of acute appendicitis.

Nausea and vomiting have been impressed upon us as being associated with appendicitis. This is the exception and not the rule. Anorexia, or loss of appetite, is more constant and more important than either nausea or vomiting. Anorexia, nausea, and vomiting are three degrees of one symptom; anorexia is the mildest form and is associated with mild distention of the appendix; nausea the middle degree, is due to moderate distention; and vomiting, the maximum degree, is found in greatly distended appendices. The most common symptom in acute appendicitis is anorexia, and if the patient states that his appetite is not altered we doubt the diagnosis of an acute appendix. Two complaints which are extremely rare in acute appendicitis are diarrhea and chills. These are probably found in less than one per cent of the cases. Constipation is the rule.

Fever is not an early finding in acute appendicitis; in fact, if present it is suggestive of peritoneal soiling. It is true that cases of acute appendicitis may have a fever of 102°, or 103°, but these are no longer cases of appendicitis; they are cases of far advanced peritonitis. Children prove the exception to this rule. If appendices could be operated upon when the temperature is below 99° the mortality would be very low.

Acute appendicitis does *not* give right rectus rigidity. Although the reverse is taught in many schools and text books, this point should be clarified. It is impossible for an individual to contract his right rectus muscle without contracting the left; therefore, when pressure is made upon an inflamed area, both rectus muscles

ontract. When only one rectus is rigid it suggests an underlying mass, such as a tumor or abscess. When both recti contract to pressure it should be considered "muscular defense" rather than right or left rectus rigidity. The importance of this bears emphasis when we realize that diagnosis, treatment and prognosis may depend upon the presence of right rectus rigidity or simple muscular defense.

The iliopsoas and obturator signs are not signs which diagnose acute appendicitis, but rather locate an acute appendix. Probably a misconception has arisen because these signs are usually discussed under the heading of acute appendicitis; they may, however, be produced in other diseases. The right iliopsoas sign is elicited by placing the patient on his left side and hyper-extending the right leg. If positive, pain is produced over the iliopsoas fascia which will be manifested in the region of the right lower quadrant. In the presence of a history of acute appendicitis this would signify that the inflamed appendix is overlying the iliopsoas fascia and is retrocecal. A positive obturator sign will locate an inflamed pelvic appendix. It is conducted in the following way: with the patient on his back the thigh is flexed upon the abdomen and the leg upon the thigh; the leg is then abducted. This causes internal rotation of the thigh and stretches the obturator internus muscle. If this produces pain it is diagnostic of a fasciitis involving the obturator fascia, which could be caused by an inflamed tube, appendix, ovarian cyst, etc. If the patient elicits a history of acute appendicitis with a positive obturator sign, we conclude that the appendix is low-lying and in the pelvis. Rovsing's sign is also helpful. It is elicited by pressing over the sigmoid, the colonic gas which has been pushed to the right will produce pain over the cecal region; this is quite diagnostic of acute appendicitis.

Routine bi-digital examinations are done; at times an acute appendix or appendiceal mass may be felt. Late and neglected appendices may produce a pelvic abscess which points rectally or vaginally, and this examination reveals the proper site and time for incision and drainage.

The laboratory data usually consist of a white blood count and a urinalysis. More important than the white blood count or urinalysis is a differential blood count; this is easy to do and is more accurate. If the "poly" count is high, it is assumed that an acute infectious process is present; a high "poly" count in the presence of a low white count means a poor prognosis. The urinalysis is usually negative but may be misleading; a few red cells in the urine are not pathognomonic of renal pathology. Negative urines have been recorded where a renal stone completely blocks the ureter so that no blood or pus can pass into the bladder.

The reason for the high mortality in acute appendicitis can be summarized in the alliteration utilizing the three "P's", namely, Purgation, Procrastination and Poor Surgical Judgement.

Acute Cholecystitis

The dictum that certain types of people are predisposed to certain types of diseases seems to be correct. The gallbladder type is described as fair, fat and forty, usually being a female in the latter third or fourth decade and somewhat obese. There is always an exception to the rule, hence, the most fulminating hydrops of the gallbladder on our service was seen in a young, thin boy of sixteen. The age of forty is related to a previous history of pregnancy, and this is theoretically explained in the following way: the average female has her children in the second decade of life and while pregnant she develops a physiologic hypercholesterolemia. Some of this cholesterol deposits on the mucous membrane of the gallbladder, forms polypi which break off and become the nuclei for stones. It may take from ten to twenty years for gallstones to attain any appreciable size, so that by the time she reaches her fourth decade the stone is large enough to obstruct or irritate. Nulliparous

women can also have gallstones or gallbladder disease, but this too, is the exception and not the rule.

The history of recurrent attacks of abdominal pain in a middle aged female, so severe that the physician must administer a sedative, is an acute gallbladder until proved otherwise. Acute appendicitis does not require morphine; renal colics will be differentiated presently, and coronary occlusion is rare in the female. One of the most unusual lesions noted in the female is a perforated peptic ulcer. The gallbladder patient also presents a previous history of "selective dyspepsia." By this we mean that there are certain specific foods that she cannot tolerate. There are four primary offenders to these foods: they are fried and fatty foods, raw apples, cucumbers and cabbage. The patient does not use the term "dyspepsia," but describes this distress as the two "B's": namely, bloating and belching. To summarize and describe the gallbladder patient one may use an alliteration and state that she is the patient with the seven "f's": she is the Fair, Fat, Fertile, Flatulent, Flabby, Female of Forty.

The complaint is one of pain, and it is important to determine the type of pain which is present. A constant pain is due to edema, but colicky pain is caused by obstruction. This is one of the factors which indicate whether the case should be treated conservatively or surgically. It is unwise to treat an obstructed lesion conservatively since these are cases which result in early gangrene and perforation. Morphine should not be used in gallbladder disease because it is a smooth muscle contractor, and since the gallbladder is a smooth muscle organ one should not administer a medicament which would stimulate its activity. By increasing muscle tonus, morphine may actually aggravate or provoke gallbladder pain and colic. One should not state, however, that the drug must never be used in gallbladder disease since it still has its place, namely, to prevent shock. These patients are treated first with nitrite therapy. One breaks an amyl nitrite bead and lets the patient inhale the vapors; $\frac{1}{100}$th grain of nitroglycerin is placed under the tongue, and 3 grains of sodium amytal or any other barbiturate is given by mouth. If this gives no relief we administer a hypodermic which consists of 100 mg. of demerol and $\frac{1}{100}$th of a grain of nitroglycerin. Should these measures fail, antispasmodic therapy with such drugs as papaverine, aminophylline, et cetera, is tried. Morphine is used only after all other measures have failed.

Gallbladder pain is usually located under the right costal margin, but may be referred to the stomach since these two organs originate from the same embryologic segment. The stomach responds to this stimulus in one of these types of gastric spasms: (1) pylorospasm, (2) midgastric spasm and (3) cardiospasm. If a pylorospasm is produced the gallbladder condition might be confused with peptic ulcer; if midgastric spasm results, a stomach carcinoma may be erroneously diagnosed; and if associated with cardiospasm, the pain appears on the left (pseudo-coronary pain) and coronary disease may incorrectly project itself into the diagnostic picture.

Referred pain should not be confused with radiation of pain. By radiation we mean that gallbladder pain, located under the right costal margin, may radiate along the path of the seventh intercostal nerve to the inferior angle of the right scapula, or the interscapular region. Gallbladder pain, therefore, cannot radiate to the right shoulder. Shoulder pain is an entirely different mechanism which involves the phrenic nerve and is indicative of peritonitis. When a gallbladder patient has true shoulder pain a diagnosis of gangrenous or ruptured gallbladder with biliary peritonitis should be made.

Temperature, pulse and respirations are included under the heading of physical examination. The patient with an acute gallbladder has an early high fever, hence, a temperature of 102° is not unusual within the first twelve to twenty-four hours of acute cholecystitis. The early fever

is explained by the absence of a submucosa. Since this tough resisting layer is lacking, there is greater chance for early contamination and absorption in the peritoneal cavity. The patient has a pulse which is increased according to the temperature, therefore, for every degree rise in fever there will be approximately a ten beat increase in pulse rate. Respirations are slightly increased because breathing is painful. This is due to the fact that the inflamed gallbladder rubs against the sensitive parietal peritoneum; because of this, acute gallbladder disease may be confused with pneumonia or pleurisy.

Although pain, a symptom, may be referred anywhere along its nervous path, tenderness, physical finding, remains at the site of pathology. This is an excellent diagnostic rule having few if any exceptions. The tenderness of gallbladder disease will be located in the region of the right costal margin. If it is most marked on a level with the umbilicus, it may be difficult to determine whether the condition is an inflamed, low-lying gallbladder or an acute high-lying retrocecal appendix. Two ways aid in the differentiation of these two conditions. First, we recall that the normal abdomen reveals a tympanitic note to percussion in all four quadrants. If the tenderness opposite the umbilicus is due to an inflamed gallbladder, we assume that the organ is unusually large or that a ptotic liver with an inflamed gallbladder at its free border is present. This would cause an obliteration of the normal tympany in the right upper quadrant and in its place the percussion note would be one of dullness or flatness. If the patient presents tenderness on the level with the umbilicus and retains normal tympany in the right upper quadrant, this would point to a high-lying retrocecal appendix. Another method of differentiating the gallbladder and appendix is by means of Ligat's test. This test locates areas of hyperesthesia over an inflamed organ. If the tenderness is due to gallbladder disease, an area of hyperesthesia (elicited by picking up the skin and letting it drop) is present from the umbilicus upward to the right costal margin. If the tenderness is due to an acute appendix, the area of hyperesthesia will be found from the umbilicus down to Poupart's ligament.

A rectal examination is done as a routine in every physical examination. More important than the rectal or vaginal examination is a so-called bi-digital, which is conducted by placing the index finger in the vagina and the middle finger in the rectum with the perineum in between. This will immediately orient the examiner and adnexal pathology will be revealed.

A flat roentgenogram should be taken in every acute abdominal condition. One may determine whether a calcified gallbladder or visible stones are present. It also gives an indication as to whether or not the liver is enlarged or ptotic. Routine laboratory tests are done.

Perforated Peptic Ulcer

This condition is rare in females. Usually a previous history of peptic ulcer or hemorrhage can be obtained, but the onset may be with perforation.

The patient states that he was seized with a sudden pain, usually after eating; this was so severe that it doubled him up. The classical picture of perforated peptic ulcer with board-like rigidity and a shock-like syndrome is too well known to bear repetition. Two signs which should be sought for in every case, however, are: (1) the findings with auscultation and (2) the presence of a pneumoperitoneum. Auscultation reveals an absolutely silent abdomen when an ulcer perforates, leaks and soils the peritoneal cavity. This is not new, since the late J. B. Murphy stressed the importance of this finding many decades ago. When intestinal sounds are present, the diagnosis of perforated peptic ulcer is remote. There are exceptions, and one of these will be discussed presently under the subject of forme fruste ulcer. The next sign which helps clinch the diagnosis is the demon-

stration of a spontaneous pneumoperitoneum. Normally a magenblase or stomach air bubble is present. When an ulcer perforates, this air bubble escapes into the general peritoneal cavity, and can be demonstrated either by percussion or with the fluoroscope; the latter is by far the more accurate. The patient is placed on his left side so that the free air bubble may gravitate upward between the liver and the right hemidiaphragm. By so doing, the liver is displaced downward and is separated from the diaphragm. Normally, the liver hugs the diaphragm and no air space is visible between them. If this air is of an appreciable amount, normal liver dullness is obliterated and in its place a tympanitic note is produced by percussion. The sign is easy to demonstrate, quite pathognomonic of perforated peptic ulcer, and present in about seventy per cent of all cases.

The forme fruste ulcer deserves special mention. The term refers to a pin-point perforation in the stomach or duodenum which is immediately sealed over by muscular contraction or by the overlying liver. Therefore, the spillage is minimal and the amount of peritoneal soiling is small. Such patients may experience a sudden sharp pain in the epigastrium, but the typical physical findings are lacking. The patient may be able to straighten up and walk about. Abdominal sounds are usually present and the air bubble may remain intragastric, having had no chance to leave the small perforation. These patients, therefore, present a misleading picture and have been misdiagnosed. However, with the ingestion of their next meal they usually reperforate and then present the typical findings.

The temperature, pulse and respirations will depend upon whether or not shock is present. Most perforated peptic ulcers present a shock-like picture which varies in its intensity. The shock associated with perforated ulcer responds rapidly to therapy. Within a few hours, the classical picture of peritonitis develops with the associated increase in temperature, pulse and respiratory rate.

The contents from a perforated ulcer may pass downward along the so-called "paracolic gutter of Moynihan," pool around the appendix and produce exquisite tenderness at McBurney's point. The diagnostician must then be on his guard, since such a history would suggest an epigastric distress with localization to the right lower quadrant which could be confused with an acute appendix. Upon exploratory operation, free fluid will be found in the peritoneal cavity with all signs of a peritonitis, and a red and injected appendix seen and removed. These patients usually die if the leaking ulcer is overlooked. This catastrophe can be avoided if, before closing the abdomen, the appendix is opened and the mucous membrane examined. Since acute appendicitis starts in the lumen of the appendix and travels outward, a normal appearing mucous membrane would suggest looking elsewhere for the cause of the peritonitis.

Laboratory data include the flat roentgenogram which has been discussed under the subject of spontaneous pneumoperitoneum. Routine blood count and urinalysis are done. Some of these patients might have bled, and although perforated ulcers are known not to produce massive hemorrhage, signs of a secondary anemia may be present.

Acute Hemorrhagic Pancreatitis

It is important to recall that this disease may appear in one of two forms: either acute edematous pancreatitis or hemorrhagic pancreatitis. The former presents a mild clinical picture, but the latter which is associated with fat necrosis and occasionally a hemorrhagic peritonitis produces a fulminating one. The acute edematous form usually improves rapidly with therapy within 48 hours, but hemorrhagic pancreatitis gets progressively worse and often requires surgical intervention. It is the hemorrhagic type, therefore, which is important to identify and treat promptly.

Although the etiology of pancreatitis is unknown, there seems to be a mechanical factor

which is associated with Spasm, Stones, Swelling and Stasis. Recent work seems to emphasize the relationship between acute pancreatitis and acute cholecystitis. This seems to be due to a common factor which is an obstruction distal to the junction of the pancreatic and common bile ducts converting them into a "common channel." An actual reflux of pancreatic juice into the gallbladder during an attack of acute pancreatitis has been shown. The patient who develops acute pancreatitis is usually of the same type that develops gallbladder disease, therefore, the condition is more common in females, rarely occurring before the age of forty, and is seen in stout people. The ratio of colored to white is 1 to 50. The attack usually follows the ingestion of a heavy meal. The pain is dramatic, sudden and excruciating; it is felt in the epigastrium, and radiates into one or both loins. In this way pancreatic pain radiation resembles an inverted fan. When the patient sits up or lies on his abdomen, the pain is relieved, and is aggravated when he is on his back. Hence, in most pancreatic conditions, be they tumors or inflammations, the patient is usually found lying on his abdomen or in a sitting position. Reflex vomiting or retching almost always occur; emesis which is truly reflex in nature is never feculent.

Physical examination reveals a patient who is usually in shock with cold and clammy extremities, subnormal temperature, and a rapid, thready pulse. Local epigastric tenderness is almost always present and is associated with a type of muscular defense which is localized to the same area. The rigidity is not truly board-like in nature, and the tenderness is most marked midway between the umbilicus and the xiphoid. An occasional finding is ecchymosis in one or both loins, or at times around the umbilicus. This is due to extravasated blood which finds its way around the retroperitoneal space and presents itself as greenish yellow or purplish discolorations. This finding, however, takes two or three days to appear. Mild jaundice is present in about half of the cases; this is explained by the fact that the common duct is pressed upon by a swollen head of the pancreas. Abdominal auscultation usually reveals a quiet but not silent abdomen.

Laboratory findings may be helpful in the diagnosis. An increase of serum amylase is suggestive in the acute phase, although a normal reading does not rule out acute pancreatitis. Polowe, has emphasized the importance of determining the blood amylase activity in terms of cuprous oxide precipitation. He has shown that moderate to marked blood amylase activity is almost always associated with disease of the pancreas, and normal or decreased blood amylase almost always excludes pancreatitis. Hypocalcemia is usually present and the level found is usually below nine. A flat roentgenogram of the abdomen may reveal a separation of the upper and lower limbs of the duodenum brought about by an edema of the head of the pancreas. This latter finding is unusual.

Renal Colics

Stones are not the only substance which produce renal colics, since the same syndrome may be produced by a small blood clot, inspissated pus, uratic debris, or a kinking of the ureteropelvic junction in a ptotic kidney.

The condition is more common in males, and the patient may reveal a history of previous attacks, a hereditary influence, a story of gout, or parathyroid pathology.

The patient complains of a sudden pain which starts in the lumbar region and radiates to the testicle, vulva or the inner aspect of the thigh. With this pain he becomes extremely restless and thrashes about. A patient who is experiencing a colic is restless and moves about, but one who has a peritonitis lies perfectly quiet and resents being moved. Vomiting is a common symptom, as is a frequency of urination. During the act of micturition the pain may be altered.

Physical examination rarely reveals any eleva-

tion in temperature, but extremely characteristic of the condition is a bradycardia. It has ofttimes been stated that when a patient with an acute abdomen has "a clean tongue and a slow pulse" he has a renal colic until proved otherwise. Tenderness is most marked in the region of the twelfth rib of the involved side, and to elicit this finding it is unnecessary and cruel to utilize any type of "punch" test. The tenderness is so exquisite that mild percussion will demonstrate it. We prefer to use the term "Murphy tap" to "Murphy punch." A zone of hyperesthesia is usually found posteriorly at the level of and slightly below the twelfth rib. If this area is anesthetized with novocaine, the hyperesthesia and pain disappear.

A flat roentgenogram may reveal a stone if such is present, but this is not reliable since non-opaque substances may also produce kidney colic. An intravenous pyelogram can be made without disturbing the patient, and if necessary, the films can be taken at the bedside with the aid of a stationary grid. The significant finding for a diagnosis of a stone in the ureter is the anuria which may be present on the affected side; the opposite side shows normal excretion. The kidney on the affected side usually appears increased in density since the dye in these tubules is more concentrated. This finding is sufficient for diagnosis of non-opaque stones in the ureter. A catheterized specimen of urine usually reveals pus, blood and albumin. The presence or absence of pus and blood in the urine is not pathognomonic since a stone may completely block the ureter and result in a normal urine. On the other hand, an inflamed appendix may be attached to the ureter, kidney or bladder, resulting in a secondary ureteritis, nephritis or cystitis with an associated hematuria. In such instances the laboratory report may actually be misleading.

Coronary Disease

Although this belongs to the realm of the internist, the general practitioner as well as the surgeon must be on guard to avoid the fatal error of confusing an acute coronary disease with an acute abdominal condition.

Men are most susceptible to coronary disease, and it is found usually in those past the age of forty. A previous history of dyspnea or pain in the chest during exertion or excitement may be elicited. The attack is sudden, with severe pain in the chest which radiates out the left arm or toward the abdomen or both shoulders. There is a sense of impending death with severe fright which usually supersedes the complaint of pain. The radiation may be toward the epigastrium, so that the examiner's attention is directed to the abdomen rather than the chest. A usual complaint during such an attack is one of "indigestion." Although the pain of acute coronary disease may occur in the abdomen, it does not become localized, hence, no area of local abdominal tenderness is ever found. Marked abdominal distention may be present in coronary pathology, but muscle defense or rectus rigidity are lacking. In abdominal catastrophes the patient lies perfectly quiet, but the coronary patient resembles colic in that he is restless and tosses about. The acute cardiac patient presents veins in the neck which are distended and full, in contrast to the patient with the surgical abdomen who may appear pale and bloodless. Signs of impaired circulation are usually present, such as dyspnea, orthopnea, and cyanosis. Auscultation will usually reveal rales in both bases due to pulmonary congestion. Cardiac enlargement, feeble heart sounds and occasionally a pericardial friction rub may be found. During auscultation of the abdomen, normal intestinal sounds will be heard which are absent or diminished in cases of spreading peritonitis.

It should also be emphasized that a distinction should be made between acute coronary occlusion and acute coronary insufficiency. Acute coronary insufficiency presents itself as an episode of angina pectoris varying from simple

transitory chest pain or pressure to severe pain or collapse. It is usually relieved by glyceryl-trinitrate. Coronary occlusion is the complete closure of an artery and is not influenced or precipitated by external environmental factors. It is not relieved by glyceryl-trinitrate, which may even aggravate it.

Positive electrocardiographic findings are pathognomonic, but one is not always fortunate enough to have an electrocardiogram handy. Leukocytosis may be present some hours after the disease takes place, and the urine is usually negative unless there is associated renal pathology.

It is realized that many other conditions at times require differentiation in the acute abdomen, among them strangulated hernias, regional ileitis, mesenteric lymphadenitis, mesenteric thrombosis, ruptured ectopic pregnancy, ruptured graafian follicle, ileocecal tuberculosis, vasitis, torsion of the omentum, volvulus, intussusception, etc., etc., ad infinitum. However, when one misses one of the unusual conditions he does not feel quite as responsible or guilty, as he would having missed one of the forementioned "Big Six."

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SYMPOSIUM ON DIABETES*

GENERAL MANAGEMENT

J. SHELDON EASTLAND, M.D.

There are about 1,000,000 known diabetics in the United States and from spot surveys conducted over the United States it is estimated that there are another 1,000,000 people who have diabetes and are unaware of it. Hence the duties of the physician are two-fold: 1) treating the known diabetics, and 2) finding the unknown. The problems of detecting the unknown diabetic has been greatly intensified by the untiring efforts of the American Diabetes Association and various medical societies. A year 'round detection drive is encouraged and annually one week is set aside as Diabetes Week in which concentrated efforts are brought to play in order to increase the physicians interest and at the same time to get people to have an annual survey. These efforts bring out individuals who have few or none of the symptoms of the disease. By the early diagnosis the disease usually may be easily brought under control and many distressing symptoms and complications may be avoided.

Tonight, however, we are primarily interested in the treatment of the known diabetic. Hospital treatment, although often desirable, is by no means always essential. The diabetic patient who cannot go to the hospital need not be denied the benefits of modern treatment, for the fundamentals may be easily mastered with a little assistance from the physician. The education of a diabetic and his family is always most essential to success in treatment.

Objectives of Treatment are several:

1) To keep the urine sugar free and the blood sugar normal. Recent observations have proven that hyperglycemia per se is the factor responsible for progressive islet damage in the pancreas. It has been demonstrated that this islet-cell damage may be prevented or

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reversed by means of proper dietary measures in conjunction with insulin.

2) To relieve symptoms.
3) To restore and maintain normal vigor and optimal body weight.
4) To enable the patient to share as completely as possible in the normal enjoyment of living. (I think this is so often overlooked.)

The basis of treatment today as in past years is the diet. However, new discoveries and changing viewpoints have profoundly influenced the type of diet advocated for the diabetic. Divergence of opinion makes it almost impossible that any single type of diet will meet with universal approval. *The fundamental principle is to avoid overfeeding.* The diabetic diet is a quantitative diet and this principle implies accurate control of the intake of food. Whether the food should be weighed or merely measured by ordinary kitchen equipment (measuring cup, tablespoon, etc.) is a matter of opinion. The diet should be palatable and satisfying both in quality and quantity. It must supply correct caloric requirements, contain proper proportions of carbohydrates, protein, fat, vitamins, and minerals. The caloric value of the diet is calculated on the patient's normal or ideal weight in Kilograms. For resting state the total calories needed can be calculated by multiplying Ideal Weight by 20. Moderate activity—multiply Ideal Weight by 30, and Strenuous activity—multiply Ideal Weight by 40. Having obtained the patient's caloric requirement the next step is to determine the number of grams of Carbohydrate, Protein, and Fat.

Example Assuming Patient's Ideal Weight is 70 Kilo.

Weight (70 Kilo.) × 30 (Moderate Activity) = 2100 Total Calories

Protein = 1 gram per Kilo (70) = 70 grams × 4 = 280 Calories

Carbohydrate + Fat (Calories) = 2100 minus 280 = 1820 Calories.

Carbohydrates = 1820 ÷ 2 = 910 Cal. ÷ 4 = 227 grams

Fat = 1820 ÷ 2 = 910 Cal. ÷ 9 = 100 grams

The task of converting grams of Carbohydrate, Protein, and Fat into terms of actual food has been greatly simplified in the "Diabetes Guidebook for the Physician" published recently by the American Diabetes Association.

This simplified method of diet calculation is based on the idea of food exchanges. The common foods allowed are divided into six groups. (Milk, Vegetable, Fruit, Bread exchanges, Meat exchanges, and Fat exchanges). In each of these groups are listed the kinds and amounts of food that have approximately the same nutritional value in carbohydrate, protein, and fat. For example, when one serving of fruit is allowed in the diet any of the fruits listed in the fruit exchange may be used in the amount specified for one serving. By the use of these food groups it is possible to calculate diet prescriptions with considerable ease. The patient is thus benefited by a diet that is made to suit his food habits along with a great variety in foodstuffs.

Nutritive adequacy of the diet is assured by including the same basic protective foods that are recommended for the normal individual—

Milk	1 pt. (adults)
Meat, fish, fowl, eggs, and cheese	4–5 ounces
Whole grain or enriched cereal or bread as prescribed	
Fruit (one citrus fruit or tomato)	2 servings
Vegetables—oen green or yellow	2 servings
Butter or fortified margarine	as prescribed

These foods should be calculated into the diet first and the remaining protein, fat, and carbohydrate may then be distributed as desired among the allowed foods. This simplified method of diet calculating is a procedure to insure the diet is adjusted to the food preferences and special requirements of the individual. "Diet lists"

prepared in advance can be used for convenience provided they are modified to suit the special needs of the individual. (There are 6 sample diets (ADA) of different caloric value.)

"Specialty" foods are not recommended. They are expensive. The information, if any, of the package may be confusing and misleading. They are not needed for the diabetic can usually eat all the natural foods in the proper amounts.

Once the diet has been prepared the patient is instructed to proceed with his usual activities. I might add at this point that we are considering the ambulatory patient with uncomplicated diabetes although many of the same principles are applicable to the hospitalized patient. Diet is the first step in treatment. The second step is the qualitative urinalysis for sugar. The performance of this test by the patient at home is absolutely essential to the proper management of his disease. There are two most commonly used methods for the collection of the urine specimens—1) Four fractions daily—urine voided (a) between breakfast and lunch, (b) between lunch and dinner, (c) between dinner and bedtime, (d) between bedtime and breakfast. 2) Single specimens voided before meals and bedtime.

The use of Benedicts qualitative solution for sugar testing probably gives the most dependable results. The Clinitest tablet method is by far the most convenient and is sufficiently reliable if the reagent is fresh. A written record should be kept of each analysis and brought to the physician each visit. If the daily specimens show much sugar a quantitative analysis of a portion of the 24 hour collection of urine should be made, or blood sugar determinations can be carried out.

If any one specimen continues to show a large amount of sugar rearrangement of the diet may be tried.

When the diet, designed to correct abnormal body weight or maintain normal body weight, fails to render the urine essentially sugar free after two to three weeks the use of insulin is indicated.

The third step in treatment is the administration of insulin. Probably about 65% of the diabetic patients will require the use of insulin. Today there are four preparations of insulin commercially available. Regular or Crystalline, NPH, Globin, and Protamine Zinc insulin, differing clinically in their rate and duration of action.

The action of regular insulin is rapid in its onset reaching its peak of action about 2½ hours following injection. After about 4½ hours it rapidly begins to lose its effectiveness. Protamine Zinc onset of action is slow and the peak is not reached until about 16 hours. Then there is a slow gradual loss of effectiveness. Hence, this insulin has little value in emergencies. It is probably the insulin of choice in the mild elderly diabetic. Globin and NPH have an intermediate place as far as rate of onset and duration are concerned. At the present time I believe that NPH insulin is the most ideal preparation available.

When a single dose of insulin (regardless of type) is given the most advantageous time is before breakfast. With regular insulin probably more than one injection daily will be necessary. In the case of Protamine Zinc one daily injection will usually control the average mild case. In more severe cases a second injection will probably be necessary, and then it is best that regular insulin be given at the same time of the Protamine Zinc. By this means the glycosuria appearing during the day can be controlled.

If NPH insulin is used it is best to begin with 10–20 units given before breakfast. The dose can be increased about 4 units every three days until the urine specimen on arising or particularly a later afternoon specimen becomes sugar free. If sugar persists only in breakfast-lunch specimen about 5–10 units of regular insulin can be mixed with morning dose of NPH.

Time does not permit further details in insulin management. In conclusion, the objectives of treatment of diabetes are best attained when the blood sugar level and glycosuria are strictly controlled. This is best accomplished by measured diets and insulin.

JUVENILE DIABETES

HARRIET G. GUILD, M.D.

Symptomatology and pathological physiology are the same in the child as in the adult, but the problem as a whole is, in many respects, quite different. The features of the disease and its management that are peculiar to the child are the subject of this discussion.

1. In the child we see the disease in pure form, unmodified by contributing factors such as obesity, arteriosclerosis or life long abuse of diet.

2. Although there may be a long latent period, the disease is of congenital origin and the defect, as a result of which the child is destined to have diabetes, is inborn. Once the diabetic state comes into being, however, the development of the full blown disease picture is much more rapid than in the adult.

Example: A 2 year old child seen by us in 1948 had accidentally had a glucose tolerance test done for other reasons, with results that were within normal limits, 2 weeks before polyuria was first noted. Admitted to the hospital 3 weeks after the first complaint, the child was on the verge of acidosis with a fasting blood sugar of 300 mg.% and a typical diabetic glucose tolerance curve with blood sugar still over 400 mg.% after 5 hours.

3. Hereditary factor is important and becomes increasingly apparent the longer the patient is followed. For example, statistics regarding family history may indicate a positive history in one out of five at the time of onset, yet in the same group of patients re-surveyed after several years, the incidence of a positive family history may become as high as 3 out of 5.

Example: In one of our patients with a negative family history at onset at the age of 2, there is now, after 20 years, a positive history which includes two 'teen age maternal siblings, a younger brother and a cousin.

4. In the child the disease is always potentially severe, even though in its incipience it may seem mild. We speak of the mild *stage* of diabetes rather than of mild *cases* of diabetes. In the pre-insulin days, a child with diabetes could get along fairly well with dietary control for 6 months or a year, but thereafter it was a losing game and death usually occurred within 2 or 3 years. Since the discovery of insulin has made survival possible, we find that there is a steady increase in the insulin requirement during the first two years of the disease, after which a level is established in terms of ratio of insulin dosage to total available carbohydrate in the diet which is fairly well maintained. Hence we know that any child with diabetes will require insulin, however mild the disease may appear during the first few months.

5. The dietary requirement of the child has to allow for growth and development as well as maintenance of body weight. Calculations are, therefore, approached from a different angle and the requirement is ever changing as growth proceeds.

6. Regulation and maintenance of control in the child are complicated by his greater lability, with reference to:

a) The frequency and severity of infections
b) The ease of production of acidosis
c) Degree of activity from day to day, with effect of exercise on his metabolic state.
d) Insulin reactions

7. The problem of diabetes in the child is

always a dual problem in that one must work with both mother and child. The first hurdle is the mother's acceptance of the disease and the fact that the child will never again be without it. Furthermore, the child, who is the patient, is not initially responsible for himself.

8. In the child himself, the pattern is ever changing. Problems differ from one individual to another, according to the age and temperament of the child and may differ in the same child at different age periods. In general the younger the child at onset, the more easily is training accomplished.

9. In the child, as in the adult, vascular complications may appear after ten or more years of disease. Because of the absence of other complicating factors, however, the relationship of these to the diabetes itself is in many instances more clear cut, and cause and effect perhaps more readily perceived.

When all of the above factors are taken into account, it becomes clear that the management of diabetes in the child is an educational process starting with the onset of the disease and continuing until the child reaches full adulthood. It concerns not only the teaching of the facts of diabetes with regard to diet and insulin, but even more, the guidance and training of a human being to a certain way of life. This educational process has as its goal the production of a normal well-adjusted individual who has learned to live with a disease that is always with him and to handle it intelligently in a way that permits smooth sailing but at the same time allows a minimum of inconvenience. Since the vascular complications referred to above seem to be less frequent and less severe in those in whom good control has been consistently maintained, it involves also the acceptance of the importance of careful regulation.

Thus, regulation of the diabetic child becomes a stepwise process, in which the first step is the calculation of a diet that is optimal for normal growth and development, with the use of scales for accuracy in measurement. After the diet is calculated, an amount of insulin is given that will make it possible for the child to utilize the diet. No effort is made to avoid the use of insulin even during the mild, early stage of the disease. Such a diet should provide 60–80 calories per kg. of body weight in 24 hours, throughout the period of active growth. The carbohydrate quota averages 100–200 gm. daily (35–50% of the total calories); the protein 2½ to 3 gm per kg. of body weight; and the fat supplies the remaining calories. Usually the carbohydrate is evenly distributed among the three meals, with a small portion between meals and at bedtime, and the elements of the diet are such as comprise the general family menu.

As mother and child become oriented with regard to the basic procedures, and become familiar with the use of scales and the composition of various foods, instruction is given in substitutions and equivalents and the inclusion of special foods that the child may particularly desire, with emphasis on the importance of a well balanced diet and just passing reference to the things to be avoided.

Liberalization in the quantity of foods that do not materially alter the carbohydrate content of the diet is the next step. At this stage only the foods of high carbohydrate content are measured accurately. Finally, after several years of this regime, as good dietary habits have become established, and the disease with its hazards is clearly understood, the scales are discarded, since now the patient is prepared to select, more or less automatically, a diet that is properly balanced and suited to his needs and one that is relatively constant in carbohydrate content from day to day. It is desirable, however, at this stage to check the accuracy of estimates by careful measurement of all foods on one or two days each month.

With regard to the type of insulin used, it is usually more satisfactory to start with crystalline or regular insulin, because of its greater flexibility as compared with protamine and because most children during the mild stage can be regu-

lated easily on two doses a day. When two doses a day no longer suffice for satisfactory regulation, a shift to protamine is made. This involves the use of both crystalline and protamine (given simultaneously before breakfast), since, at this stage, childhood diabetes cannot be controlled by protamine alone. NPH insulin has not proved very satisfactory in our hands since it also usually involves the necessity for additional crystalline insulin in the morning and often fails to control nocturnal glycosuria without a supplemental dose of insulin at supper time as well. It does, however, have its place in special cases and is most useful in those patients in whom the diabetic status has become stabilized after growth has been completed.

To achieve stability in regulation, it is desirable to permit a measure of glycosuria since insulin reactions are thus more easily avoided and allowance is then made for the variations in the child's activity. A spill of 20 gm. of sugar daily is within the limits of satisfactory regulation.

Careful regulation of the disease throughout childhood, by steps such as those outlined above, and the establishment of good habits with reference to it are essential to the child's future welfare, both physical and psychological. Furthermore, the understanding and achievement of good control creates self assurance and makes it possible for the individual to follow with confidence whatever course he chooses in study, work and even marriage.

DIABETIC EMERGENCIES; PRE AND POSTOPERATIVE TREATMENT

BENJAMIN F. JONES, M.D.

This evening I have been assigned the topic of diabetic emergencies. While there are many critical situations arising in the treatment of diabetes, the basic emergency is the development of acidosis with coma, actual or impending. Recent advances in the treatment of diabetic acidosis and coma are concerned with three topics, viz., (1) the use of insulin; (2) criteria for the diagnosis of diabetic coma and guides for therapy; (3) changes in the metabolism of potassium.

With the introduction of insulin, a dramatic fall occurred in the percentage of deaths due to diabetic coma. Further reduction in the death rate thereafter was achieved more slowly. Although clinicians knew the value of insulin in daily management of the disease, they were fearful of hypoglycemia and unaware of the decreasing effectiveness of insulin with the advance of ketosis. Today it is well recognized that insulin given in the early stages of acidosis is many times more effective than the same amount administered after the progress of acidosis and the development of coma. Cases of diabetes exhibiting great resistance to insulin have been discovered and studied. The analogy with these conditions has led many to speak of "increased insulin resistance" in keto-acidosis, although no one really understands the decreased effectiveness of insulin in any of these states. The need for large initial doses of insulin early in acidosis, however, has been clearly shown, and this constitutes a great therapeutic advance. So too is the recognition of the need for large amounts of insulin in the early hours of treatment when more extreme degrees of keto-acidosis are present.

The schedule of insulin dosage used in the treatment of diabetic acidosis and coma has been based on various criteria, including the depression of carbon dioxide content of the plasma, the elevation of the blood sugar, the urine content of sugar and acetone bodies, and the combination of these interpreted in the light of the over-all clinical picture. Joslin and his associates (1) advise the use of the blood sugar level and the depression of the carbon dioxide combining power of the plasma as guides to

therapy. They recommend initial injection of 100 units of regular insulin subcutaneously for adults, if blood sugar exceeds 300 mg. per 100 c.c. and if the blood CO_2 content is 9 millimols per liter (20 volumes per cent) or less. In cases with blood sugar between 600 and 1000 mg. they would give 300 units as an initial dose and with a blood sugar of 1000 mg., they would give 400 units initially. The value of the blood sugar concentration and the carbon dioxide combining power of the blood as guides for the administration of insulin has been questioned. Certainly they are of little or no value if glucose or alkali have been used in the previous therapy.

A very useful guide to insulin therapy would seem to be the concentration of acetone bodies in the blood, including β-hydroxybutyric acid. As a routine measure, this procedure is not practical because of the time and special techniques needed for the quantitative estimation of the acetone bodies of the blood. Many years ago it was demonstrated that the Rothera qualitative test for aceto-acetic acid and acetone could be applied directly to the blood plasma. In some clinics this method in conjunction with blood sugar and plasma CO_2 levels has reportedly been used for some time as a guide to therapy in diabetic coma. It is surprising that this method has not been more widely used. So far as I know it has not been used extensively in any Baltimore Clinic. Recently Duncan (2) has reported his experience with the qualitative determination of aceto-acetic acid and acetone by this method as an aid to the early diagnosis of diabetic keto-acidosis and coma and as a guide in the treatment of coma. Ketonemia is graded as 1 to 4 plus by the Rothera test in the same way as in tests of the urine. For clinical purposes, criteria for the diagnosis of diabetic coma have been established as glycosuria and a 4 plus reaction for acetone in the blood plasma. As a guide for the dosage of insulin in coma, the acetone test is applied to undiluted plasma and to successive dilutions of plasma with normal saline to 50%, 25%, 12.5% and 6.25%. When only the undiluted plasma shows a 4+ acetone reaction, 100 units of regular insulin are given as the initial dose. A 4+ reading also at 50% dilution is the signal for an initial dose of 200 units of insulin; and the persistence of 4+ reading at 25% and 12.5% dilution calls for initial doses of 300 and 400 units of insulin respectively. In like manner the degree of ketonemia estimated in this way is followed as a guide for insulin administration in the course of further treatment. The test can be quickly carried out on a few drops of plasma obtained by allowing freshly drawn oxalated blood to stand for a short time. Commercial acetone test powders or tablets such as are used in qualitative tests for acetone in the urine may be satisfactorily used in place of the usual Rothera method. This rapid and simple procedure promises to be an important aid to all physicians concerned with the care of diabetes, whether in the home, or the office, or the hospital.

The discussion of emergencies in diabetes would be incomplete without consideration of the part played by potassium. Beginning with the original report of Holler in 1946, a series of studies has established the syndrome of potassium deficiency in diabetic acidosis as a new clinical entity. Weakness of the muscles of the extremities and of respiration progressing to paralysis and respiratory failure has been noted, with onset 2 to 12 or more hours after the start of treatment for diabetic acidosis. The clinical picture is accompanied by characteristic electrocardiographic changes and a reduced serum potassium level. Treatment with potassium salts gives prompt relief of symptoms.

During the progress of keto-acidosis, there is a great loss of both potassium and sodium from the body. In spite of this loss of potassium the homeostatic mechanisms of the body assisted by dehydration and frequently by some degree of renal failure may suffice to maintain the level of serum potassium at normal or above. In this phase, deaths have been reported of patients in diabetic coma whose electrocardiogram has shown the characteristic changes of potassium

poisoning of the heart, and whose serum potassium has been found to be above the critical level of 10 milliequivalents per liter. Treatment with insulin, intravenous saline solutions and glucose solutions (though the latter is not always involved), in time depresses the potassium level below the normal of 5 milliequivalents per liter (19.5 mg.%). The critical level for the development of the electrocardiographic and clinical changes of potassium deficiency is regarded as 3 milliequivalents per liter.

There is a progressive effect of low serum potassium on the electrocardiogram.* As the serum potassium is lowered, prolongation of the QT interval is the first change seen in the electrocardiogram. The upper limit of normal for the QT interval is roughly one-half the R-R interval and both of course will vary with the heart rate.

The second change noted is lowering or inversion of the T wave and in the third stage the depression of the ST segments is seen. The final stage shows a depressed take-off of the ST segments with a low delayed T wave. The first stage may be seen at serum potassium levels of 3 milliequivalents or less. The final stage is seen with potassium levels in the range of 1.5 milliequivalents per liter or lower.

In like manner progressive electrocardiographic changes with elevation of the serum potassium may be demonstrated. The first change seen is the elevation of the T waves and this is followed by a decrease in the amplitude of the R wave. The third stage is characterized by auricular arrest and P waves are absent. At higher levels the ST segment becomes depressed and the QRS duration increases. In the terminal stage with potassium levels in the range of 9.5 to 10 milliequivalents per liter the form of the electrocardiogram becomes completely changed with biphasic curves occurring in completely irregular fashion. The level at which electrocardiographic changes are first noted with high serum potassium is approximately 6 milliequivalents per liter. Obviously some of these electrocardiographic changes are non-specific, but the patterns are characteristic and readily identified. There appears to be no correlation, however, between the level of the potassium in the serum and the degree of electrocardiographic abnormality in many cases (4).

Replacement of potassium poses a nice problem in therapy. The situation is always complex. There is no routine treatment. Experience indicates that certain procedures are relatively safe and effective on the one hand, while certain precautions are essential on the other.

The indications for giving potassium are

(1) reduction of serum potassium to 3 milliequivalents or lower
(2) characteristic electrocardiographic changes diagnostic of potassium deficiency
(3) development of weakness in the muscles of respiration and in the muscles of the extremities (recognition of the clinical syndrome of potassium deficiency)
(4) when there is a history of prolonged or severe acidosis, starvation, debility, vomiting, diarrhoea, or prolonged gastrointestinal suction, —by way of prophylaxis.

If the condition of the patient permits, potassium should be given by mouth. Three grams of potassium chloride dissolved in a pint of appropriate fluid (soup, vegetable juice or milk) provides about one half the daily average adult dietary intake of potassium. One hundred and fifty c.c. of this solution hourly (equivalent to about 1 gram of potassium chloride) can be safely given when renal function is adequate and continued till the serum potassium is within normal limits.

In urgent cases and where oral administration is impossible, parenteral treatment is necessary. Potassium chloride can be added to a liter of 5% glucose solution in amounts from 1.5 to 6 grams, giving solutions ranging in concentration from about 20 to 80 milliequivalents of potassium per liter. These solutions can be given intravenously

* See the diagrams prepared by Dr. R. S. Ross and published by Drs. J. E. Howard and R. A. Carey in the Journal of Clinical Endocrinology (3).

at a rate adjusted so that 5 to 10 milliequivalents of potassium per hour (and not more than 10 milliequivalents per hour) are run in, regardless of the concentration of the solution. Parenteral treatment should be controlled by reference to frequent electrocardiographic tracings and serum potassium determinations. It should be continued until oral administration of potassium is possible or until the serum potassium is brought within normal limits. Very large quantities of injected potassium may be necessary to achieve this result, up to 30 or more grams of potassium chloride in 24 hours.

Potassium should not be given orally or parenterally if urinary output is impaired. Potassium should not be given parenterally unless the indications are definite and the serum potassium is known to be depressed. As a rule, potassium is not needed until several hours after insulin treatment has begun and the blood sugar begins to fall significantly. Whenever a patient is receiving potassium intravenously, means should be available for rapid treatment with calcium gluconate and intravenous glucose. Patients receiving potassium intravenously should never be left unattended.

I turn now to a more cheerful subject, the preparation of diabetics for surgical operations and their postoperative care. The aim of preoperative care is to secure a normal state of nutrition, and particularly normal carbohydrate metabolism. Adequate stores of carbohydrate in liver and muscles are a defense against ketosis and the stress of operation. So also are normal reserves of water and electrolytes and vitamins. Ideally all diabetics scheduled for elective or non-emergency surgery should be admitted to the hospital for a period of days or weeks before the operation. In these days of crowded hospitals and operating room schedules, an adequate period of pre-operative care is often difficult or impossible to arrange. In the case of diabetics, however, it pays heavy dividends in better tolerance to surgery, and in superior results of surgery, to say nothing of shortened convalescence time. We should not forget that this type of preparation can be done in the home in many cases. An attempt should be made to reduce the weight in obese patients, and often this is unusually successful when they face the prospect of operation. The value of rest and supportive care for a generous period of time is strikingly seen in the case of diabetic patients in the older age group and in those with hypertensive cardiovascular disease, malnutrition and general debility. All diabetics scheduled for elective or non-emergency surgery should be admitted to the hospital in time for routine diagnostic procedures.

For purposes of convenience these patients may be divided into three groups:

Group I

Patients whose diabetes is controlled by diet without insulin and who are able to take a diet of normal caloric content including at least 150 grams of carbohydrate. These patients require only general measures in preparation for operation. They are given no insulin or glucose preoperatively, and in the post-operative period are given insulin only as needed to control glycosuria and ketosis.

Group II

Patients who are able to avoid taking insulin by restriction of carbohydrate below 150 grams daily or who are taking 150 or more grams of carbohydrate with small amounts of insulin (up to 15 units daily). Patients in this group should be placed on a diet of 150 grams of carbohydrate, if not taking this much or more. If the patient is already taking insulin, the dose may be increased by addition of regular insulin to the PZ or NPH insulin to take care of the added carbohydrate. Patients not taking insulin should receive small amounts of NPH insulin for this purpose. This procedure can be carried out in the home in the week before operation.

Group III

Patients requiring more than 15 units of insulin in order to tolerate a diet normal in calories

and with 150 grams carbohydrate or more. In this group also may be placed those patients ordinarily belonging in the other two groups, but whose condition is unfavorable because of infection or other disease. Patients in this group will profit especially by an adequate period of hospitalization prior to operation. This is obligatory when there is keto-acidosis or badly controlled diabetes. Patients with milder degrees of keto-acidosis may be treated with half the usual dose of insulin given as protamine zinc, with supplements of regular insulin as needed. The majority of patients in this group will need preoperative hospitalization for study and treatment of conditions other than diabetes, chiefly cardiovascular disease, renal disease, urinary tract obstruction or infection. Recognition of the need for restriction of metabolites (sodium and protein, e.g.) preoperatively may prevent much grief during and after the operation in this type of patient. Except in the case of patients showing frank acidosis, it is usually neither necessary nor desirable to abandon the long-acting insulin (PZ, NPH or globin insulin) in favor of frequent doses of regular insulin. Diet and insulin can usually be adjusted in a reasonable time so that the fasting and postabsorptive blood sugars approximate normal and the urine is free of ketone bodies. A moderate amount of glycosuria is permitted (10 to 15 grams in 24 hours).

Emergency operations on the diabetic can be carried out in the way outlined above and without delay or special treatment, if the patient is not in ketosis. Special care in following these patients should be exercised when their cases are unfamiliar to the attending physicians or the diabetes has been previously unrecognized. The diabetic in ketosis or coma should have vigorous treatment, while operation is postponed, till acidosis and dehydration are corrected. Antibiotics, blood transfusions, and other measures may be needed to offset the delay. Ether anesthesia should be avoided in emergency cases.

Anesthesia. The preferred anesthetic for a diabetic is the one producing the least disturbance of carbohydrate metabolism and the acid-base balance. Local or regional block anesthesia and spinal anesthesia thus should be preferred over general anesthesia. Among general anesthetics, the preferred anesthetic should be the one producing the least anoxia, the least tendency to acidosis, and the least gastrointestinal disturbance. Ether and chloroform have been shown to produce extreme hyperglycemia and acidosis. Liver glycogen is severely depleted by ether anesthesia. The requirements of the diabetic state, however, may not coincide with the demands of the surgeon and compromises must be made. Ether should be used cautiously with patients whose diabetes is controlled preoperatively and is contraindicated in keto-acidosis. Otherwise the choice of a general anesthetic should be made on the same basis as in surgery on the non-diabetic. I know of no specific contra-indication to curare unless it should happen to be used when the patient is in the hypokalemic state. Minimal doses of morphia preoperatively have been advised in diabetes because of the danger of anoxia. In my experience the tolerance of diabetics to the drug is not significantly less than that of non-diabetics.

On the day of operation a special diabetic chart is started in which will be recorded insulin, intravenous glucose and other solutions, oral feedings, blood sugar, urine sugar and ketone bodies and other significant data.

If antibiotics are being given for infection, consideration should be given to increasing the dose before operation and to changes from oral to parenteral administration after the operation. Prophylactic administration of appropriate antibiotics is indicated in diabetics before operation, especially in operations on the urinary and gastro-intestinal tract.

Operations upon diabetics should be scheduled at an early hour in the morning. Whatever the hour of operation the patient should receive an injection of insulin before operation, not later than the customary time for taking insulin. This preoperative insulin should be given in the form

of protamine zinc, because of the stabilizing effect of its prolonged action. Judicious use of protamine zinc insulin may greatly facilitate negotiation of the crisis of operation. Some recommend preoperative injection of ¼ the previously required insulin in this form, others as much as one half. In my experience ⅓ the amount of insulin previously taken as PZ, or 10 units, whichever is the greater, has proved satisfactory. The important point to recognize is that every diabetic taking insulin who undergoes surgery will need an injection of insulin before going to the operating room. It is equally important to appreciate the fact that such patients will require on the day of operation as much total insulin as they were taking previously, or more.

To avoid hypoglycemia from the continuing effect of the PZ insulin taken the day before the pre-operative injection, intravenous glucose should be given. One liter of 5% glucose (50 grams glucose) should be given to patients who have received 25 units of PZ insulin preoperatively and 750 cc. to 1000 cc. of 10% glucose (75–100 grams glucose) to those receiving more. The infusion should be started just after the insulin is injected, and the rate of flow so adjusted that a substantial portion of the fluid will still be available when anesthesia is started. If the operation is long delayed, the infusion can be given in two portions, the latter starting at about the time of induction of anesthesia.

Various plans have been followed in the administration of insulin, intravenous fluids and nourishment in the postoperative period. It should be emphasized that no such plan can be safely or effectively applied without regard for the demands of the individual case and close observation of the course of events. There are many pitfalls in this jungle, and he who has more up-to-the minute data on the progress of the case and awareness of what may happen will tread with more confidence.

For the uncomplicated case, the following plan is usually effective: Total carbohydrate of not less than 150 grams is given on the day of operation and at least 3 liters of fluid. One liter of this should be in the form of normal saline, unless sodium restriction is necessary. In this case the amount is adjusted to the situation. The amount of carbohydrate given is intended to equal or exceed moderately that in the usual diet, including the portion derived from protein. The additional carbohydrate required is given in intravenous infusions beginning shortly after the end of operation and repeated every 4 to 6 hours. With the first infusion of 50 grams or more of glucose, a dose of insulin equal to about ⅓ of the estimated remaining required dose for the day (based on the patient's previous insulin requirements, and not less than 10 units) is given subcutaneously. The urine is tested every two hours for sugar and acetone by convenient rapid test methods. (Clinitest sugar and Denco or Acetest acetone tests). In this way the insulin dose is set for the next period, the aim being to avoid ketosis on the one hand and glycosuria on the other. As soon as the patient is able to take oral nourishment, liquid and soft foods are presented in feedings at regular meal times approximating his usual diet. Any deficit in intake is made up, if significant, by further intravenous glucose. PZ insulin is given daily each morning before nourishment without interruption. On successive days the patient is restored to the previous diet and insulin is adjusted by gradually increasing the PZ fraction while decreasing the regular or CZ insulin. Before discharge the patient may be shifted to NPH insulin or kept on PZ insulin alone, or supplemented by regular insulin.

In some cases the problem of postoperative care becomes extremely complicated. This is particularly apt to occur when there is much impairment of renal function, emesis or diarrhoea, and when the operation involves the gastro-intestinal tract extensively, necessitating prolonged periods of total intravenous feeding. The removal of gastro-intestinal fluid by suction also produces complex difficulties in diabetic postoperative management. In general, when nutri-

tion is entirely or mostly by the intravenous route for a period longer than three days, it is necessary to add protein hydrolysates and soluble vitamins to the infusion fluids. A standard preparation of protein hydrolysate as the 5% solution in glucose or saline, can be given in amounts sufficient to provide the equivalent of at least 0.5 gram protein per kilogram of body weight per day. One liter of such an hydrolysate contains nitrogen approximately equivalent to 40 grams of protein. The more dilute the mixture is and the more slowly it is given, the better it will be tolerated, as intravenous administration of protein hydrolysate solution causes phlebitis. The protein hydrolysate can be given in one infusion of 8 hours or in two equal portions over a period of four hours each. Patients who have lost fluid and electrolytes by nausea, vomiting, or suction should be specially studied to determine the amounts of fluid and electrolytes needed for replacement. A special modified electrolyte solution such as that devised by Darrow or Butler containing sodium chloride, potassium, magnesium, phosphate and glucose may be used where replacement of large amounts of electrolytes is necessary. For a short period of time the situation can often be handled by addition of potassium chloride alone in amounts up to six grams daily to the saline and glucose infusions. Where total intravenous feeding with extensive replacement of protein is necessary, the patient should have special balance studies, and will fare better if placed in a ward with facilities for more elaborate metabolic studies. In patients nourished by total intravenous feeding, the management of diabetes is often a minor aspect of the total problem.

When patients exhibit unexplained ketosis postoperatively, search should be made for infection. Sometimes the onset of ketosis heralds the fact that amputation of a toe has been unsuccessful in stopping the progress of atrophy and infection. When ketosis sets in after patients have resumed oral feedings, inadequate intake of carbohydrate is to be suspected and the ketosis should be vigorously treated with additional regular insulin and forcing of carbohydrate by mouth or vein. Conversely patients who have been relieved of gangrenous and infected tissue by operation, may rather suddenly pass into hypoglycemia in the days just after the operation. In one such case, the patient was able to discontinue the use of insulin postoperatively altogether although taking about 60 units daily before the operation.

Thanks to insulin and better understanding of diabetes, thousands of diabetics are today undergoing operations successfully following a course little different from the non-diabetic. With adequate care, the mortality rate in diabetics undergoing surgery need be no greater than that in similar operations upon non-diabetics. Sprague and Wilder in 1939 at the Mayo Clinic noted 2 hospital deaths (1.8% mortality) in 109 diabetic patients undergoing cholecystectomy for cholecystitis. In the same period, 12 hospital deaths occurred amongst 703 non-diabetic patients subjected to cholecystectomy (1.7% mortality).

There are few emergencies in medicine which call for greater versatility and alertness in treatment than keto-acidosis, actual or threatened, in the diabetic, as a complication of surgery. Nothing could be more disastrous than to attempt to follow a formula or routine in these cases. The foregoing remarks are not intended as such, but rather to outline the general principles of management. The special problems presented by each case must be solved as they arise.

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PERIPHERAL VASCULAR DISTURBANCES

GEORGE H. YEAGER, M.D.

Dr. J. Sheldon Eastland: Our last speaker is last in position on the program but surely is not last in the diabetic field. I think everyone who has handled any diabetes at all has been troubled with the peripheral vascular disturbances, and tonight, Dr. Yeager is going to go over some of them with us.

Dr. George H. Yeager: I think one of the greatest advantages of being on a panel of this type, is that you can look at the audience and see a gradual cerebral obtunding come over everyone. For that reason I am going to be purposely brief. I imagine we can gain a great deal more by interjecting a few points of controversy and then approach the problem of questions. I'm sure my predecessors will welcome questions and discussion.

I would like to preface my remarks with a statement that any patient presenting himself with a suggestion of a peripheral vascular disturbance should be screened for the possibility of co-existing diabetes. Frequently a patient will present minor complaints with reference to an extremity, and very often the possibility is overlooked that the symptoms may be an early manifestation of diabetes. Within a short period of time the patient may develop a rather serious infection or tissue necrosis before it is determined that the problem is that of a diabetic who may be out of control.

The subject tonight is Peripheral Vascular Disease in its components and relationship to diabetes. I am not going to discuss infections except to state that infection in the diabetic should be treated as potentially of serious consequence. Such patients should be put at bed rest and the infection should be controlled before normal physical activities are permitted.

The majority of patients from the peripheral vascular viewpoint, if they are a known diabetic, have the psychological handicap of knowing that they have a disease that is going to be with them the rest of their life. Possibly they have noticed weakness in their extremities, with limitation of ability to walk. It is serious to them because they wonder if they are going to be confronted with the end of their economic self-sustaining life.

Many patients will probably tell you that they have noticed a declining exercise tolerance for a period of months or years but didn't want to discuss it because of the possibility of jeopardizing their employment.

After ascertaining that there is a lowering of exercise tolerance, and finding evidence of organic deterioration in the vascular bed, it becomes important to evolve various lines of therapy. The first approach should be a discussion with the patient as to his general outlook.

Quite often we have a tendency to minimize the implications. Many times these patients are told simply to "repace" themselves; learn to walk more slowly and to cut down their activities. Many times they are told to give up tobacco without stressing its importance.

A patient presenting definite subjective and objective evidence of vascular disease should be screened from a number of viewpoints. The basic viewpoint should be to determine how much organic obliteration is present and how much secondary vasoconstrictive gradient is associated with the condition. By such evaluation a therapeutic approach can be developed.

With the popularity of sympathectomies the tendency probably is to sympathectomize too many patients. It is a good procedure. However, there are certain patients definitely harmed by sympathectomy. Individuals with marked stasis and loss of tonicity in their vascular bed are not going to be helped by a sympathectomy and probably are going to be harmed.

Many patients, with conservative care of their extremities and a definite regime of sympatho-

lytic therapy, will increase their exercise tolerance and avert the necessity of a sympathectomy.

Surgery should be reserved for the group that can be promised improvement. Sympathectomy from the viewpoint of increasing exercise tolerance will probably be disappointing. In the group on whom sympathectomy is advocated, I don't believe you should tell them if they can walk a block before a sympathectomy they will be able to walk two or three blocks after their sympathectomy. I doubt it. I doubt if there is going to be any change in their exercise tolerance.

FIGURE 1

Therapeutic Armentarium—Peripheral Vascular Disease

DRUG	DOSAGE
Ilidar	
Regitine	50 mg.
Priscoline	25 mg.
Etamon Chloride	3–5 cc.
Roniacol Tartrate	50 mg.
Bistrium (Hexamethonium Chloride)	250 mg.
Alcohol	
I. V. Procaine—0.1%	
Paravertebral Block	
Cortone?	
Vitamine E (Ephynal Acetate; Tocophorex; etc.)	
Heparin (Treburon; Depo-heparin)	
Dicoumarol	

If a sympathectomy is going to be advocated it should be approached from the viewpoint of enhancing their collateral circulation, of enhancing their general outlook, and probably retarding the progress of their deterioration; their blockage. It is hoped to keep these people active within reason and avoid an issue of complete disability.

There was a young English doctor over here recently—some of you may have seen a movie that he presented. It was very well done. There is a large vascular clinic in Manchester, England. That clinic has an extremely large series of sympathectomized patients on whom they have performed the procedure with the hope of increasing exercise tolerance. It was found in certain groups that a large percentage of them failed to demonstrate an increase of exercise tolerance. In the group failing to respond, cutting of the tendon of Achilles was performed.

They have a series of over two hundred such cases and claim that there is very little disturbance of gait. I suggest that we review some of our historical facts. It will be found that cutting of the tendon of Achilles was one method of treatment of captives to keep them from escaping. I believe that any benefits derived from cutting of the tendon of Achilles is due to slowing down of pace; their stride has been interfered with—they are bound to walk more slowly. If we can succeed in educating these patients to repace themselves, we should not have to resort to such a radical procedure as cutting of the tendon of Achilles.

In cutting the tendon of Achilles, a factor of phlebitis has been substituted because of the stasis that develops in the posterior calf muscles. A high percentage of the patients that have had such a tenotomy developed recurring phlebitis. It is not a type of procedure that I am personally willing to advocate. However, I am sure that there are going to be many surgeons start doing it with the hope of increasing the exercise tolerance of their patients.

Intra-arterial priscoline seems to be of some value in the conservative management of vascular disease. We should recognize that vascular disease, with or without diabetes, basically presents the same problem of management. After determining that their diabetes is under control, the management of the peripheral vascular disease becomes identical. Recalcitrant ulcers, with areas of necrosis seem to demarcate more promptly, and heal with the use of intra-arterial priscoline. It is injected directly into the femoral artery in fifty milligram doses.

In the past we have all been a little apprehensive about the trauma of intra-arterial injections. It has been well proven that there is no hazard to such a mode of therapy.

Other drugs will be developed that will be as

good or better than priscoline. I simply cite it as an example of the intra-arterial use of a drug compatible with more satisfactory results than has been noticed in the past.

The use of cortone probably seems somewhat contradictory. I believe that Cortone seems to be effective because it blocks out pain stimuli. I doubt if it has any effect on peripheral vascular disease, and certainly there is a great deal of evidence that it may be detrimental. We see this "blocking out" with Cortone in the toxic effects of peritonitis and toxemia.

Cortone is being used experimentally from the viewpoint that it is of value and that the exercise tolerance of these individuals can be increased.

I might interject that it is being tried in Buerger's disease. It seems to have some merit in controlling the excruciating pain of that condition.

Throughout this geographical area, the tendency is to perform a relatively conservative type of lumbar sympathectomy with extirpation over the bodies of L-2, L-3 and possibly the body of L-4. There are some groups that contend this is inadequate and that stripping should extend over L-1, and on to the 12th thoracic. They cite cases of failure of ulcers to heal with stripping of the ganglionated chain on the bodies of L-2 and L-3, and healing with a more radical operation.

We are all aware that environmental factors make a great deal of difference in our results. Many of the reported cases have been from Florida. Until a great deal more work has been done, a more extensive stripping of the ganglionated chain for a lesion in the lower extremities probably should not be widely advocated.

Dr. Eastland: The Panel has attempted to get a few of the high spots of this big subject of diabetes. I hope it has been interesting and of some value.

The hour, I know, is getting late, but if there are any questions—I see down here there is such a thing as Question Period. If there are any, I'm sure we would be glad to try and answer them. Does anyone have anything in particular? If not, we will turn the meeting back to our President, Dr. Fort.

Dr. Fort: I just want to thank each one of the members of the Panel very much for their time and effort in our behalf and I hope that maybe some time they will come back and be with us again. Thank you. The meeting is adjourned.

NICOTINIC ACID THERAPY IN VASOCONSTRICTION TYPE OF HEADACHE[1]

ZACHARIAH R. MORGAN, M.D.

Headache is one of the most common complaints encountered by a physician in daily practice. Although much has been written on headache, this subject is of sufficient interest to warrant a brief communication. In this presentation only the so-called "Migraine syndrome" will be considered.

The physiological mechanism and the many factors in the etiology of the different types of headache included in the migraine syndrome is not completely understood and there is no doubt a wide diversion of views on the cause of headaches. Although great strides have been made in the understanding of some of the etiologic factors involved and the therapeutic measures for their relief, there is still much to be known regarding this subject. Varying etiological factors have been adduced by different authors; how-

[1] Read before the American Therapeutic Society, Chicago, Illinois, June 7, 1952.

ever, Von Storck (1) has pointed out that the syndrome as a whole has a multiple pathogenesis that may be subdivided into various types in which one or another etiology is predominant. Schumacher and Wolff (2) in their experimental studies of headache, suggest that there is first a vasoconstriction of the cerebral arteries but the pain does not begin until the occurrence of dilatation of the extra-cerebral arteries, especially the branches of the external carotid artery. The latter phenomenon is the chief cause of the pain in headache. Carl Pfeiffer (et al.) (3) suggest the possibility that blood volume changes might participate in the etiology of migraine headache, and present data which they claim justify the separation from the so-called common headache into two new types, namely, relaxation headache and the caffeine-withdrawal headache. The former is accompanied by a decrease in the arterial blood volume, but the latter is attended by a relative increase in the arterial blood volume. No consistent blood electrolyte changes occur with relaxation or migraine headache. The determination of the serum proteins and hematocrit during and after migraine headache indicates that the migraine syndrome is accompanied by a relative hemoconcentration. Many other known factors in this syndrome can be correlated with changes in blood volume and peripheral vascular tone. Dreisbach (4) (et al.) corroborate the findings regarding the caffeine-withdrawal headache. Moench (5) states that in these particular individuals, with special predisposition and psychobiologic equipment that tend to create sustained pernicious emotional states and fatigue, it may be reasonable to postulate that labile physiologic mechanisms within the cranium are set off which end in the untoward chain of events constituting the attack of migraine. Wolff (6), in a psychobiologic study of forty-six migraine subjects had shown that they were so constituted as to be peculiarly prone to the development of pernicious emotional states, either sustained or with acute episodic exacerbations. In some of them a sudden increase in stress was provoked by clearly definable life situations. Such periods were associated with an increase in the intensity and frequency of attacks of migraine. He suggests that in addition to allergic, biochemical and roentgenographic studies, the personality of migraine subjects be investigated in order to determine to what extent the psychobiologic factors play in the role of their personalities.

It is noteworthy the role which nicotinic acid plays in the treatment and relief of the vasoconstrictor type of headache associated with depression. Although the use of nicotinic acid in the treatment of headaches is not new, it has not been sufficiently stressed in the treatment of this form of headache. Washburn (7) discusses the use of nicotinic acid in the treatment of certain depressed states. He points out that despite their varying properties and actions, the pharmacologic agents which have proved negligible in the treatment of schizophrenia, possess in common certain vasoconstricting effects, while those pharmacologic agents which tend to improve the condition have certain vasodilating effects. Many investigators (8, 9, 10) have directed attention to the fact that epinephrine, amphetamine, and ephedrine not only fail to help a patient in the so-called depressed state, but often seem to increase the severity of the symptoms, while cocaine, alcohol, sodium amytal, carbon dioxide inhalations and shock therapy have proved beneficial. Following the use of nicotinic acid in various depressed states, Sydenstricker and Cleckley (11) conclude that factors other than dietary disturbances play important roles—factors yet undetermined and perhaps unsuspected may also influence the failure of the neurones to receive and utilize an adequate supply of vitamins. Furthermore, Cleckley (12) believes it probable in some cases, that the beneficial effect observed after the administration of nicotinic acid is the result of vasodilatation rather than the correction of a deficiency state. Aring (13) likewise concludes that there is a marked

increase in the cerebral blood flow following the use of nicotinic acid.

Goldzieher and Popkin (14) recommended the use of Intravenous Sodium Nicotinate in the symptomatic treatment of (a) severe ecliopathic migraine, (b) migraine and (c) post-spinal tap dephalalgia. Cachin and Beaumont (15) obtained favorable results in fifteen of the cases of long standing migraine following the use of nicotinic acid or sodium nicotinate.

It occurred to me that the use of nicotinic acid should be employed in those cases which are of the emotional or tension type of headache where Ergotamine or Dihydroergotamine (Gynergen) has failed to give relief, and which at times often increases the severity of the pain. Attention must be drawn to the fact that the pain is probably caused by vasoconstriction of the cerebral arteries rather than dilatation of the extra-cerebral arteries. So it seemed opportune to recommend this form of treatment for the vasoconstriction types of headaches associated with depression so that others may afford some relief to their patients in this variety of migraine. This paper is therefore primarily concerned with a group of patients who are not relieved by the usual drugs utilized for the treatment of headaches and whose headache is due to vasoconstriction. Five cases are reported which show favorable results over a long period. Although the number of cases comprised in this study is small, the clear-cut clinical picture of relief obtained in this group warrants further study of the vasoconstriction type of headache associated with depression.

Method of Treatment

Intravenous Administration: Nicotinic acid was initially administered by the intravenous route to six patients. In all instances this was immediately followed by oral administration. A solution of nicotinic acid 100 mgm. in 10 cc. of sterile water was injected intravenously usually at the rate of 20 to 30 drops per minute. The rapidity of injection was determined, however, in each case by the degree of flushing and warmth which ensued, as well as any evidence of hypersensitivity or untoward reactions to the drug. The time required for the administration often varies from 10 to 20 minutes. The intravenous therapy was continued as follows: Three weeks at twice a week, three weeks at once a week, and once every other week for three doses. At the conclusion of the first treatment the patient left the office, and was given a prescription for two hundred 50 mgm. tablets of nicotinic acid together with a typed schedule of instructions for its oral use; the first dose to be taken preceding the next meal.

Oral Administration: The schedule for oral administration is as follows: (a) For the first two days, three 50 mgm. tablets are to be taken at 15 minute intervals preceding each meal. Example—breakfast hour 8:00 A.M., first tablet at 7:15 A.M., the second tablet at 7:30 A.M., the third tablet at 7:45 A.M. (b) If no untoward reaction, such as headache or nausea occurs, the dosage is to be increased on the third and fourth days as follows: four 50 mgm. tablets are to be taken before each meal, according to the fifteen minute schedule, the additional tablet being added to the first and second dosages. Example—100 mgm.—50 mgm.—50 mgm. (c) On the fifth and sixth days five 50 mgm. tablets are to be taken before each meal, the additional tablets being added to the first and second dosages. Example—100 mgm.—100 mgm.—50 mgm. (d) On the seventh and eighth days six 50 mgm. tablets are to be taken at the 15 minute schedule before each meal, two tablets being taken together. Example—100 mgm.—100 mgm.—100 mgm. Thus by the eighth day the patient was receiving a total of 900 mgm. a day. The patient was maintained on the maximum dosage for a period of one week to 10 days at which time the dosage was decreased in the same order as it had been increased. The average duration for the administration of nicotinic acid thus varied from periods of two to six weeks after which most patients were given 100 mgm. three times a day.

The rapidity of the initial relief following the use of nicotinic acid was always in proportion to the intensity of the vasomotor reaction characterized by the bright red flushing of the face, rapidly spreading over the entire trunk, tingling of the fingers and of the lower extremities and accompanied by a marked sensation of heat. After the second or third injection these side reactions gradually became less severe. All of the patients were most cooperative and there was little or no complaint about the side reactions.

One must give the drug slowly at first but fast enough to get a definite flush. So far as I have been able to find after a rather exhaustive search of the literature there seems to be no contraindication of its use.

CASE REPORTS

Case I, G. K., Age 62. Present illness began about 2 years ago, being characterized by severe headache beginning in the occipital region and neck and spreading over the head into the frontal region. Attacks last from one hour to twenty-four hours and gradually become more intense.

During the past year these attacks have become much worse and occur once or twice each week. He does not complain of any other symptoms. There is no history of dizziness, nausea, or temperature elevation.

Patient had been seen and treated at one of Baltimore's best clinics, and also examined by a psychiatrist and neurosurgeon.

Positive Findings on Physical Examination:

1. Somewhat overweight elderly white male, who did not appear ill.
2. Left testicle had been surgically removed several years before—cause unknown.
3. Evidence of mild chronic prostatitis.
4. Moderate osteo-arthritic changes in the cervical spine.

All other laboratory and x-ray examinations were negative. Ergotamine did not affect the headache. At times, sodium amytal and large doses of bromide would abort the attack.

A provisional diagnosis was made of chronic tension state accompanied by depressive features and periodic vascular head pains; Hypertrophic arthritis of cervical spine (mild); Chronic prostatitis.

The patient was given nicotinic acid as described above which has given him marked relief from the symptoms. When contacted six months ago, he was still taking 100 mgm. before each meal and he had been free of symptoms for 16 months except for an occasional mild pain which is relieved by aspirin.

Case II, Mrs. K. C., Housewife age 58, began having dull aching pain on left side of head, which in past year became so severe at times that it was necessary for her to lie down for several hours. For past three or four months these attacks had been coming on about every three or four days, and sometimes lasted from twelve to twenty-four hours. They were accompanied by dizziness, nausea and vomiting which did not afford any relief from pain.

She had been told that her trouble was due to hypertension and had had several months of treatment without relief.

Her past history is not important except that as a child she had trouble with her right hip and was put in traction for several weeks. X-ray disclosed a "flat socket" with ankylosis. She has had no discomfort for years except for mild aching when there is a change in weather. Mobility is limited.

Positive Findings on Physical Examination:

1. Somewhat overweight white female who did not appear ill.
2. Hypertension: 192/88 at rest; 210/84 two minutes after exercise.
3. Moderate osteo-arthritic changes in fingers, knees and right hip.
4. Electrocardiogram and other cardiac findings were negative.

A diagnosis was made of (1) Chronic tension state with periodic vascular head pains, (2) Mild hypertension, (3) Mild osteo-arthritis.

Patient was given Ergotamine which almost immediately caused severe head pain accompanied by nausea and vomiting. She was then started on the nicotinic acid treatment as outlined and responded with amazingly good results.

She now takes 100 mgm. three times a day and has had marked relief from symptoms. Her blood pressure now ranges from 160 to 180 systolic and 90 to 84 diastolic.

I have followed her closely for a period of one year.

Case III, Mrs. J. G., Housewife, 38 years old, had always had headache on the fourth and fifth day of menstrual period since puberty. At first, headaches were mild but gradually became more severe. For past three years, they had been coming on at any time irrespective of menstrual cycle. She was usually awakened with a headache which lasted for about 24 hours, and it was accompanied by photophobia, nausea, vomiting and a sense of depression. She has been married for several years and has two children.

Patient was treated by a psychiatrist for nine months without relief. One physician diagnosed her trouble as hypertension. She had been given ammonium chloride before her menstrual period and calcium at the onset of her period. Ergotamine seemed to intensify headache.

Positive Findings on Physical Examination:

1. Somewhat overweight female who does not appear ill.
2. Mild elevation of blood pressure: 142/96 at rest; 160/86 two minutes after exercise.

There is a soft apical systolic murmur which is transmitted to the anterior axillary line. There is some accentuation of A_2. She was begun on a nicotinic acid regime and for past year has had no further trouble. Blood pressure when last seen was 132/84. Patient is now taking 100 mgm. three times a day.

Case IV, Miss E. B., Age 45, minor executive in department store, complained of intermittent sick headaches for several years which had become worse over a period of past two or three years. Patient was usually awakened in the night by onset of headache which often lasted for two to twelve hours, and was followed by a sense of depression. These headaches were not relieved by Ergotamine, but were sometimes helped by aspirin. Three years before, she had been given subcutaneous histamine in an effort to "desensitize" her to this drug, but this gave her no relief. Psychiatric treatment was next tried which seemed to afford relief for a few months but the headaches then returned more severe than ever.

Physical examination and all laboratory findings were negative.

She was given nicotinic acid therapy about a year ago, and is still taking 100 mgm. three times a day. During this period, the patient has been entirely symptom-free; as expressed by her "I have a sense of well-being and for the first time in many years life seems worth living."

Case V, Miss F. B., Age 32, Restaurant hostess, began having severe frontal headaches which radiated into back of the head and often lasted throughout the day.

The headaches were always worse before menstruation and lately had become so severe that she had been unable to work. This was followed by a sense of depression.

Physical examination and all laboratory reports were negative except for a mild secondary anemia.

Ergotamine made the headache much worse. Ammonium chloride and a salt poor diet premenstrually did not seem to improve her condition.

She was given nicotinic acid therapy as outlined above and is now taking 100 mgm. three times a day. For the past six months she has been symptom-free.

Discussion

My interest in this problem has been stimulated by the experience with a small group of patients who have received many forms of therapy for their long standing intractable headaches associated with a depression, and in whom it was

possible to try the nicotinic acid therapy recommended here. The method in which the nicotinic acid treatment was administered is another point of interest worthy of clinical notice.

The general outline of therapy as recommended by Washburne (7) and in addition, the combined intravenous route with the oral route and the use of a stronger solution of nicoitnic acid was carried out in all of these cases.

Attention is called to the fact that each of these patients was first given Ergotamine by hypodermic and in four cases the pain was made much worse. In the other case, while there was no increase in the intensity of the headache, there was also no relief. Three of the cases had been given psychiatric treatment with no result. All had been treated by other physicians with many and various remedies.

While the above cases are too few and of too short a duration to draw any final conclusions, the results are encouraging. These cases set forth with particular clarity the effect of nicotinic acid therapy as outlined in this presentation. Although casual references have been made in the literature regarding the effect of nicotinic acid in the treatment of headaches, it might be of interest to point out that the usual oral therapy is often insufficient and generally leads to a stoppage of the treatment because of poor results. Furthermore, it should be noted that Ergotamine, histamine, and psychiatry have not solved the problem and that in nearly all cases the headache is accompanied by a sense of depression.

Conclusions

1. Five cases of tension headache accompanied by more or less depression are presented.

2. The method of treatment of giving nicotinic acid intravenously and combined with sustained oral nicotinic acid therapy as outlined in this communication is highly important in order to obtain good results.

3. The vasodilating effect of nicotinic acid serves to strengthen the concept that headache produced by vaso-constriction may be relieved by the use of this drug.

4. The favorable results obtained in this form of long standing headache gives convincing evidence of the use of the vasodilating effect of nicotinic acid.

5. The presentation of this small group of cases, demonstrating the apparent value of nicotinic acid, should stimulate further study of the use of this drug in the severe type of headache with depression.

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Component Medical Societies

ALLEGANY-GARRETT COUNTY MEDICAL SOCIETY

LESLIE E. DAUGHERTY, M.D.

Journal Representative

Dr. John K. Rozum has terminated his residence in Cumberland and is moving his family to Miami, Florida, where he will establish a practice in general surgery.

After graduating from Medical College in June, 1931, Dr. Rozum served a residency at the Lutheran Hospital (formerly West Baltimore General), Baltimore, Maryland and came to Cumberland in 1935. For three years he has been a Chief in Surgery at the Sacred Heart Hospital in Cumberland. He is a member of the American College of Surgeons, as of 1950, Southern Medical Association, Allegany County Medical Society and Medical and Chirurgical Faculty of the State of Maryland and has served as City Physician for approximately ten years.

Dr. Rozum served with the Navy, as a Lt. Commander in World War II with the Cub 17 Unit and was in action on Guam and Okinawa.

Dr. Ralph W. Ballin, 62 Greene Street, Cumberland has returned to private practice after serving two years in the United States Army. He spent most of the time in the Fitzsimon's Army Hospital at Denver, Colorado, assigned to Ward service.

Dr. Ballin came to Cumberland in March, 1949, after being associated at the Maryland Tuberculosis Sanitorium, Sabillisville, Maryland. He graduated from the University of Cologne, Germany. He is married to the former Helen Gillaland, of Uniontown, Pennsylvania. Dr. Ballin is on the staff of the Memorial and Sacred Heart Hospital and is now conducting the chest clinic in Cumberland.

The Allegany-Garrett County Medical Society held its May meeting at the Cumberland Country Club, beginning with a Dutch treat dinner.

Dr. Frank H. J. Figge, Professor of Anatomy at the University of Maryland School of Medicine, spoke on "Recent Trends in Cancer Research and Therapy."

The following article appeared in the Cumberland Sunday Times, May 10. It is an interesting story of Cumberland's Dr. F. A. G. Murray, one of the oldest living graduates of the University of Maryland and his relationship to Queen Elizabeth II, of England.

"The Amazing Story That Starts With the Birth of George Smith at Seldsden Hall, Scotland, in 1792 and Leads to Cumberland and Westminster Abbey—To which Is Added the Story of Allegany-Garrett County Medical Society's—Dr. F. A. G. Murray."

One descendant of a Scotsman named George Smith (of Seldsden Hall), born in 1792, resides in Cumberland.

He is Dr. F. A. G. Murray, a great great grandson.

Another great great grandchild of the same George Smith resides in England.

And on June 2 that great great grandchild's daughter will be crowned in Westminster Abbey as Elizabeth II, queen of England!

The interesting story of a Cumberland physician's relationship to a Queen was told in "Across the Desk" July 20, 1947. But the coronation next month of Dr. Murray's distant cousin lends new interest to the joint genealogy of a local physician and the new ruler of the British Empire!

George Smith was a modest man of considerable means but not of noble birth. Certainly he could scarcely have dreamed that 161 years after his birth, a descendant would be the reigning Queen of England and of his native Scotland.

There were two sons born to Mr. and Mrs. George Smith of Seldsden Hall.

One was Oswald Smith whose great granddaughter (Elizabeth Bowes Lyon) married Albert, Duke of York, afterwards George VI.

The other son of George Smith was Edward Peploe Smith, great grandfather of Dr. Murray.

The step-by-step descendants of Oswald Smith follow:

A daughter, Frances, married Claude Bowes Lyon, 13th Earl of Strathmore. Their son, the 14th Earl, was the father of Elizabeth Bowes Lyon whose marriage to the Duke of York was mentioned above.

When Edward VIII (now the Duke of Windsor) gave up his throne to marry a Maryland woman (Wallis Warfield), the Duke of York became England's ruler as George VI. Their daughter Elizabeth becomes the first reigning British queen since Victoria.

Now for the genealogy of Oswald's brother, Edward Peploe Smith.

A daughter, Emily Frances (note that Frances was also her first cousin's name!) married Archibald Murray, a noted London barrister, and their son, George Mosley Murray, was the father of Cumberland's Dr. Murray.

A minister of the Church of England, Dr. Murray's father came to the United States more than 80 years ago (in 1872). He died in 1919 while rector of St. Bartholomew's Episcopal Church in Baltimore.

One of the oldest living graduates of the University of Maryland (1897) Dr. Murray first practised in Garrett County; opened an office in Mt. Savage in 1903; served as a Medical Officer in World War I, and then came to Cumberland 34 years ago. He is still active.

The Cumberland physician always kept in closer touch with the Murray side of the family than with the Smith-Bowes Lyon relatives.

Upon the death of a cousin, General Sir Valentine Murray, who was in charge of transportation for the British Army in the First World War, that last survivor of his immediate family willed his military decoration and all the ancestral family portraits to his cousin here.

Among the paintings are oil portraits of Mr. and Mrs. Charles Murray, grandparents of Dr. Murray's grandfather, Archibald Murray. There is a pastel portrait of Dr. Murray's grandmother and two of her children, one of them Sir Valentine's father.

Oldest of the family pictures received from England six years ago is one of Dr. John Murray of Norwich (born in 1720) and one of the founders of the Society of St. Andrew in Norwich. A companion portrait to the one now owned by the Cumberlander hangs in the Norfolk and Norwich Hospital in England.

Dr. Murray's grandmother (Emily Frances Smith Murray) was a first cousin of Queen Elizabeth's grandfather, the 14th Earl of Strathmore.

Incidentally Dr. Murray's full name is Francis Alan Gordan Murray.

BALTIMORE CITY MEDICAL SOCIETY

CONRAD ACTON, M.D., *Journal Representative*

The proposed new Constitution and By-Laws were read at the 17 April meeting in accordance with the present Constitution. These will be mimeographed and sent to City Medical Society Members ahead of the October meeting when they will be voted on. It was but a step from the Constitution and By-Laws to a discussion of the change to be proposed in the Faculty Constitution and By-Laws at the Annual Meeting. These were thoroughly discussed and procedure about them tentatively agreed upon.

Dr. George Thorne gave the Kolson Memorial Lecture in the Hurd Hall that same evening and provided a good deal of competition. Dr. Thorne's talk was a return to his old stamping grounds and was well attended.

The Cardiology Postgraduate Course wound up successfully. The series of talks was appreciated by City Society Members. Thanks to the Union Memorial Hospital for the use of the hall. Orchids to the good lecturers.

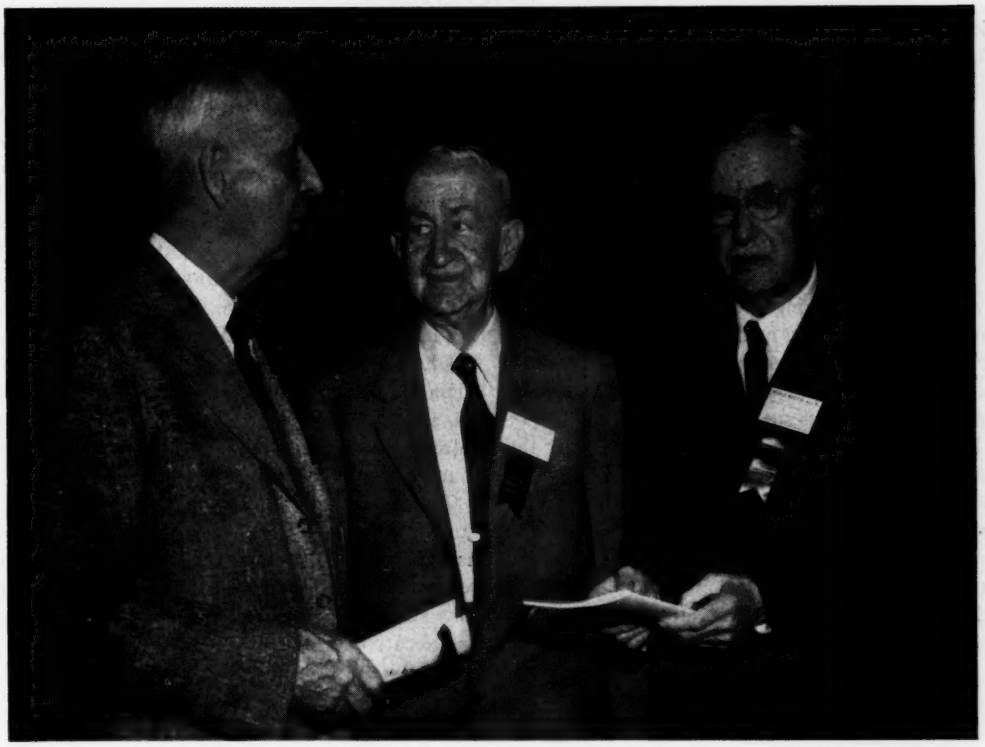
A postgraduate committee is in the process of formation. It is anticipated that postgraduate courses on other themes may be given in the Fall. If the Fall meeting is as well attended as this Spring one has been, President Fort anticipates quarterly courses on a series of major topics. The names of this Postgraduate Committee have not been released.

A joint Committee on Anesthesia Mortality has been set up. Composed of members of the City Health Department and the City Medical Society, it is conceived in the same cooperative spirit as the Joint Committee on Maternal Mortality. The Anesthetic Mortality Committee will be composed of 12 members, three at least, will be anesthetists in active practice. Ex-officios will be Chairman of the Anesthesia Section of the City Medical Society, President of the Baltimore Hospital Conference, Professors of Anesthesia at Hopkins and University Hospitals, the Chief Medical Examiner or a member of his staff and Health Commissioner of Baltimore City or his representative. The Chairman will be designated by the President of the Baltimore City Medical Society. Names are not yet available for publication but will be released probably before this goes into print.

Another new development is an outgrowth of the Faculty's Joint Committee on Medicolegal Problems. A list of Specialists in their respective fields is being prepared for the use of judges and magistrates of Baltimore City. The judicial authorities feel the need of specialized advice and support in certain problems of a medical nature.

The State Annual Meeting was held April 27, 28, 29. It has been reported in full in other sections of this Journal. It was a most delightful meeting and much credit is due the Auxiliary for the Medical Ball on Monday night and the Art Exhibit. Great work went into the preparation of these features. They gave enjoyment to many people.

A Medical Section of the City Society is finally in the process of organization. The merits and demerits of such a move have been under discussion ever since Blue Shield started. An organization meeting 12 May elected Dr. Francis Gluck, Chairman, Dr. T. Nelson Carey, Vice-Chairman, Dr. Ernest C. Brown, Jr., Secretary, and Dr. Katherine Borkovich, Treas-



DR. W. B. CAMPBELL DR. LEWIS K. WOODWARD, SR. DR. LOUIS J. HIRSCHMAN

urer. A second meeting is planned for 16 June at which time policies and committees should be decided on.

CALVERT COUNTY MEDICAL SOCIETY

PAGE C. JETT, M.D.

Journal Representative

"The new Calvert County Hospital was dedicated on May 16, 1953. This hospital, costing approximately $800,000 was completed in March 1953. It has facilities for thirty patients but can be rapidly expanded to accommodate fifty patients. This hospital was built with local and Hill-Burton funds and represents the latest in design and appointments for a one-story rural hospital with facilities for white and colored patients. All are cordially invited to visit our new hospital."

CARROLL COUNTY MEDICAL SOCIETY

75 YEAR-OLD MARYLAND DOCTOR HONORED AT RICHMOND

At First Western Hemisphere Conference of the World Medical Association, at Richmond, Va., April 21–25, three medical school graduates of 1899 compare notes on medical progress since that time: l. to r.: Dr. W. B. Campbell of Waukesha, Wisconsin; Dr. Lewis K. Woodward, Sr. of Westminster, Maryland; and Dr. Louis J. Hirschman of Traverse City, Michigan.

These 75-year-old doctors, with 45 others, were designated by the Governors of their respective states as guests of honor. The theme of the Conference was Medicine's Achievements in lengthening life and improving health. A grant to the World Medical Association by A. H. Robins Co., Inc., of

Richmond covered conference expenses. From: World Medical Association.

PRINCE GEORGE'S COUNTY MEDICAL SOCIETY

JOHN WARREN, M.D.

Journal Representative

At the April meeting, Dr. Alfred Suraci, one of our associate members, presented slides on his topic, "Problems in Reconstructive Surgery." Dr. Suraci showed us many of the new plastic materials that are now being used in his specialty.

In May we heard from Dr. Alan D. Miller of the United States Public Health Service's Mental Hygiene Clinic. Their unit, located in College Park, is one of the few experimental clinics which is still operating and serving the people of local communities.

Dr. and Mrs. Julius Kauffman had a very enjoyable cocktail party and the society was so well represented that they took the opportunity to hold an Executive Board meeting at which Dr. Wolcott Etienne was named to the Board of Directors of Prince George's Hospital. Under a new law just passed by the Maryland Legislature, the lay member selected was Mr. Ralph F. deClairmont of Mitchellville. Dr. Waldo B. Moyers, another member of the society, had already been chosen by the medical staff as its representative on the board. Dr. Moyers, who laid much of the ground work as Chairman of the Society's Legislation Committee, agreed to represent the hospital's medical staff for one year.

Dr. William Stecher, hospital radiologist, Harry Penn, hospital superintendent, and Dr. Harry Ferris, associate member in dentistry, have each given talks over Station WNAV on our program, "Community Medicine Today."

The Prince George's General Hospital's Assistant Superintendent and Treasurer, David A. McNamee, has resigned his post effective May 25. Mr. McNamee leaves to enter the Georgetown University Law School but will retain his post with this society as Executive Secretary.

150 MEDICAL GRADUATES IN ARMY'S INTERN PROGRAM

THE AMA WASHINGTON LETTER, NO. 16

A total of 150 June medical school graduates have been selected for the Army's medical intern program. All will be assigned to one of the Army's 11 teaching hospitals. The men will serve as first lieutenants in the Reserve. The largest groups, 21 each, will go to Brooke Army Hospital at Fort Sam Houston, Texas, and Walter Reed, Washington, D. C.

ADD TO LISTS OF LIBRARY AND FILMS CLEARED FOR TV

AMA NEWS NOTES, VOL. 2, NO. 5

Supplementary lists of films cleared for television and films available through the AMA's motion picture library now may be obtained from the Committee on Medical Motion Pictures. One supplement lists 38 health education films cleared for TV since publication of the original list in 1951. The other includes 12 motion pictures added to the library since the December, 1952, catalog was published.

Library

"Books shall be thy companions; bookcases and shelves, thy pleasure-nooks and gardens." *ibn Tibbon*

BOOKS ON GOUT

LOUIS KRAUSE, M.D.*

This month, we are presenting the books on GOUT. You will note that the list is large and dates back to earlier days. Gout is frequently overlooked in everyday practice; and how little do we hear of the terms of *Podagra*, which is gout in the big toe, and *Cheiragra*, which is gout in the wrist, or *Gonagra*, which is gout in the knee. In all likelihood, terms of this kind always kept the possibility of gout in the minds of the older clinicians; and for that reason, they may not have overlooked the diagnosis as frequently as we do today. Incidentally, these are the terms used by Hippocrates.

Our treatment with Colchicine really dates from the 5th century and the word gout dates from the 13th century coming from the French *Goutte*, signifying a drop and implying at the time that there was a poison that entered the joints drop by drop. The best clinical description is still the one by Sydenham who lived in the 17th century.

Today, of course, we are looking not only for primary gout, the "inborn metabolic error," but also for the secondary forms that we see in leukemia, nephritis, etc. The appended list of books in our library will prove an excellent source for information on this anciently known disease.

BOOKS ON GOUT IN THE MEDICAL AND CHIRURGICAL FACULTY LIBRARY

A Selected List

Barker, L. F. and Cole, N. B., Rheumatism; its meaning and its menace. New York, 1926.

Buckley, C. W., Arthritis, fibrositis and gout. London, 1938.

Cadogan, W., A dissertation on the gout, and all chronic diseases, jointly considered, as proceeding from the same causes. London, printed; Boston, reprinted, 1772.

Chapman, N., Lectures on the more important eruptive fevers, haemorrhages and dropsies, and on gout and rheumatism. Philadelphia, 1844.

Charcot, J. M., Maladies des vieillards, goutte et rhumatisme. Paris, 1890.

Chomel, A. F., Leçons de clinique médicale, v. 2, Rhumatisme et goutte. Paris, 1837.

Cohausen, J. H., Epistola responsoria epicritica, clarissimo et experientissimo domino Salentino Ernesto Eugenio Cohausen. Frankfurt and Leipzig, 1749.

Delpeuch, A., La goutte et le rhumatisme. Paris, 1900.

Desault, P., Dissertation sur la goutte. Paris, 1738.

Dickinson, J. C., On the tonic treatment of gout. 2nd ed. London, 1871.

Duckworth, D., A treatise on gout. London, 1890.

Duringe, M., Monographie de la goutte. 4th ed. Paris, 1830.

Ebstein, W., Die natur und behandlung der gicht. 2nd ed. Wiesbaden, 1906.

Ewart, W., Gout and goutiness. New York, 1898.

Fuller, H. W., On rheumatism, rheumatic gout and sciatica. London, 1852 and 1854, Philadelphia, 1864.

Gardiner, J., An inquiry into the nature, cause and cure of the gout. Edinburgh, 1792, Philadelphia, 1793.

Garrod, A. B., Treatise on gout and rheumatic gout. 3d ed. London, 1876.

Grimke, J., Dissertatio medica inauguralis de podagra. Edinburgh, 1808. (Thesis)

Gros, E. L., A modern pathological and therapeutical study of rheumatism, gout, rheumatoid arthritis and allied affections. New York, 1897.

Halle, J. N., Rapport sur les effets d'un remède proposé pour le traitement de la goutte. 2d ed. Paris, 1810.

Hardy, J., A candid examination on the colic of Poitou and Devonshire; with remarks on the gout. London, 1778.

* Chairman, Library Committee.

Hood, P., A treatise on gout, rheumatism and the allied affections. London, 1871.

Jones, H. B., On gravel, calculus and gout. London, 1842.

Lecorche, E., Traitement de la goutte. Paris, 1894.

Levison, F., Uric acid diathesis, gout, sand and gravel. London, 1894.

Lindsay, J., Gout, its aetiology, pathology and treatment. London, 1913.

Llewellyn, R. L. J., Aspects of rheumatism and gout; their pathogeny, prevention and control. London, 1927.

Llewellyn, R. L. J., Gout; with a section on ocular diseases in the gouty by W. M. Beaumont. London, 1920.

Longstreth, M., Rheumatism, gout and some allied disorders. New York, 1882.

Loomis, A. L. and Thompson, W. G., ed., System of practical medicine, v. 4. New York and Philadelphia, 1898.

Lucianus Samosatensis, Podagra tragice producta a Luciano. 1631. *In* Sennert, D., De arthritide tractatus. 1631. p. 109–132. Text in Greek and Latin.

Luff, A. P., Gout, its pathology and treatment. London, 1899.

Noorden, K. H. von, Technique of reduction cures and gout. New York, 1910.

Proust, A. A. and Mathieu, A., L'hygiene du goutteux. Paris, 1896.

Revere, J., An inquiry into the origin and effects of sulphurous fumigations in the cure of rheumatism, gout, etc. Baltimore, 1822. (Medical pamphlets, v. 3).

Richardière, H. A., Maladies de la nutrition, goutte—obésité—diabète. Paris, 1913.

Roberts, W., On the chemistry and therapeutics of uric acid, gravel and gout. London, 1892.

Roose, R., Gout; its relations to diseases of the liver and kidneys. 7th ed. London, 1894.

Ruhräh, J., William Cadogan (His essay on gout). New York, 1925.

Ruppaner, A., Hypodermic injections in the treatment of neuralgia, rheumatism, gout, etc. Boston, 1865.

Scot, J., An enquiry into the origin of the gout. 3d ed. London, [n.d.]

Scudamore, C., A treatise on the nature and cure of gout and rheumatism. 2d ed. London, 1817.

——, A further examination of the principles of the treatment of gout. 2d ed. London, 1833.

Sennert, D., De arthritide tractatus. Wittenberg, 1631.

Spilsbury, F., On the scurvy, gout, diet and remedy. 5th ed. Norwich, 1790.

Sydenham, T., Abhandlung über die Gicht (1861), eingeleitet und übersetzt von Julius Leopold Pagel. Leipzig, 1910.

Thomson, F. G. and Gordon, R. G., Chronic rheumatic diseases, their diagnosis and treatment. London, 1926.

Todd, R. B., Practical remarks on gout. London, 1843.

Voet, D., Dissertatio medica inauguralis de podagra, sive pedum dolore. Heusden, 1690.

Warner, F., A full and plain account of the gout. 2d. ed. London, 1768.

Wilde, P., The physiology of gout, rheumatism, and arthritis. New York, 1922.

HOW CAN THE LIBRARY HELP YOU?

The Library stands ready to serve the members in any way possible.

Do you want recent articles on the estrogenic treatment of cancer of the lung, or on eyeground changes in toxemia of pregnancy, or on some other clinical condition? Would you like Aub's original description of lead poisoning, or some other historical material? Do you need the address of a pediatrician in St. Louis, or a dermatologist in Little Rock?

The county members especially are urged to make more use of the Library. Requests by letter or telephone are answered promptly, and material is mailed out within twenty-four hours if at all possible.

Suggestions as to books the Library should purchase, and ways in which it can be more useful to members, will be much appreciated by the Library Staff.

STATE OF MARYLAND DEPARTMENT OF HEALTH

MONTHLY COMMUNICABLE DISEASE REPORT

Case Reports Received during 4-week Period, May 29–June 25, 1953

	CHICKENPOX	DIPHTHERIA	GERMAN MEASLES	HEPATITIS, INFECT.	MEASLES	MENINGITIS, MENINGOCOCCAL	MUMPS	POLIOMYELITIS, PARALYTIC	POLIOMYELITIS, NON PARALYTIC	ROCKY MT. SPOTTED FEVER	STREP. SORE THROAT INCL. SCARLET FEVER	TYPHOID FEVER	UNDULANT FEVER	WHOOPING COUGH	TUBERCULOSIS, RESPIRATORY	SYPHILIS, PRIMARY AND SECONDARY	GONORRHEA	OTHER DISEASES	DEATHS: Influenza and pneumonia
Total, 4 weeks																			
Local areas																			
Baltimore County	23	—	14	5	47	4	41	2	—	—	26	—	1	3	16	—	15	—	5
Anne Arundel	7	—	1	3	10	—	1	—	—	—	4	1	—	—	6	—	8	—	—
Howard	—	—	1	—	3	—	1	—	—	—	—	—	—	1	3	—	1	—	—
Harford	3	—	13	15	25	1	2	—	—	—	—	—	—	—	15	—	—	m-2	—
Carroll	—	—	—	1	3	—	10	—	—	—	1	—	—	—	2	—	3	—	1
Frederick	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—
Washington	—	—	—	—	3	—	1	—	—	—	—	—	—	—	7	1	3	—	1
Allegany	2	—	—	4	11	—	—	—	—	—	—	—	—	1	2	—	—	—	1
Garrett	—	—	—	10	3	—	—	—	—	—	—	—	—	—	1	—	—	—	1
Montgomery	11	—	5	5	21	1	36	1	—	—	2	—	—	—	10	—	1	—	—
Pr. George's	4	—	5	—	32	—	33	1	2	—	5	—	—	1	16	—	—	—	2
Calvert	—	—	—	2	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1
Charles	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—
Saint Mary's	—	—	—	13	1	—	—	—	—	—	—	—	—	—	1	—	1	—	2
Cecil	—	—	—	1	2	—	—	—	—	—	1	—	—	—	3	—	1	—	1
Kent	3	—	8	—	29	—	22	—	—	—	3	—	—	—	—	—	—	—	—
Queen Anne's	—	—	—	—	2	—	1	—	—	—	—	—	—	—	4	—	—	—	—
Caroline	1	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	3	—	—
Talbot	—	—	—	—	—	—	—	—	—	—	—	—	—	—	3	—	4	—	—
Dorchester	1	—	—	—	—	—	1	—	—	—	—	—	—	—	2	—	11	—	—
Wicomico	1	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	20	—	1
Worcester	1	—	—	—	1	—	1	—	—	—	—	—	—	1	2	2	—	—	—
Somerset	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	8	—	—
Total Counties	57	0	47	59	193	6	150	4	2	0	42	1	1	7	100	3	79		16
Baltimore City	65	0	34	16	148	4	225	1	0	0	107	1	0	16	124	10	553	tr-1 p-1 t-1	7
State																			
May 28–June 25, 1953	122	0	81	75	341	10	375	5	2	0	149	2	1	23	224	13	632		23
Same period 1952	332	1	106	12	405	3	90	1	—	7	79	0	1	34	177	20	612		28
5-year median	292	5	65	—	505	4	203	4		10	63	3	3	48	234	58	566		27
Cumulative totals																			
State																			
Year 1953 to date	2608	8	1370	257	1290	55	1733	11	2	2	2116	10	6	103	1212	76	3769		454
Same period 1952	2576	7	773	132	8868	60	728	8	—	7	773	8	11	124	1313	84	3211		401
5-year median	2885	47	478	—	3625	53	1355	8		16	812	12	25	357	1377	479	3275		388

m = malaria reported by Aberdeen Proving Grounds, origin Korea, home residence not stated.
p = psittacosis.
t = tetanus.
tr = trichinosis.

BLUE CROSS AND BLUE SHIELD

BLUE CROSS INTER-PLAN SERVICE BENEFIT BANK

R. H. DABNEY*

Another milestone of service was reached recently by the Blue Cross Inter-Plan Service Benefit Bank, when it paid for its 4 millionth day of patient service. Since the Bank began operations in May 1949, it has cleared more than 450,000 cases and provided payments for out-of-area subscribers of more than 44 million dollars.

The Bank is an arrangement entered into by most Blue Cross Plans in order to provide service benefits on a nationwide scale, utilizing the service contracts which each Blue Cross Plan has with the hospitals in its area. This makes it possible for a Blue Cross member, when sick while away from home, to obtain service benefits wherever he may go to the hospital. It also gives Blue Cross member hospitals a simple and reliable system for establishing credit for the out-of-town Blue Cross patient.

Each Blue Cross Plan is a separate organization, with its own subscription rates and benefits geared to local needs. Prior to the existence of the Bank, it was not possible for a Plan to give service benefits to its subscribers when hospitalized out of their area, and these cases were usually handled by providing a cash indemnity toward the hospital bill. In many cases, hospitals had the problem of getting in touch with a large number of Blue Cross Plans to determine what allowances to provide.

Here is the way the Inter-Plan Bank operates.

* Executive Director of Maryland Hospital Service, Inc. and Maryland Medical Service, Inc.

A subscriber from Baltimore is hospitalized in Chicago. On presentation of his membership card, he is admitted just as though he were a member of the Chicago Plan, and the hospital notifies that Plan of the admission. Chicago then sends a coded telegram to the Maryland Plan asking for confirmation of membership and eligibility for benefits. Maryland replies within 24 hours.

The Chicago Plan then approves the admission to the hospital and payment for the care is handled just as though the subscriber were a member of the Chicago Plan. The case is then cleared through the Inter-Plan Bank, which debits and credits each Plan's account in the Bank according to a predetermined formula related to the cost of care in Chicago and the cost of care in Maryland. Accounts in the Bank are balanced every three months.

While all Plans make every effort to transfer subscribers when they move to another area permanently, there still are a large number of subscribers who need hospital care when they are on vacation or travelling. Each month an average of over 18,000 subscribers are admitted to hospitals outside their own Plan areas, and receive Blue Cross service benefits through the Inter-Plan Bank.

Today, the Bank is indispensable to Blue Cross Plans and enables them to render better and more service to their subscribers. It has extended the service benefit principle of Blue Cross beyond the area served by each Plan, thus benefiting both the subscriber and the hospital.

REPORT FOR THE BOARD OF TRUSTEES OF MARYLAND MEDICAL SERVICE, INC.[1]

HUGH J. JEWETT, M.D., *President*

Maryland's Blue Shield Plan has come a long way in its two brief years of existence. It now serves 197,000 Marylanders, 37,000 more than at the end of 1951. Our enrollment gain for 1952 under the standard program was 50 percent, and nearly half of this gain came after September 1, when changes in the fee schedule and subscription rates materially improved the attractiveness of the Plan. The Plan's income in 1952 went over the million dollar mark and of this amount, 83.6% was paid in benefits to subscribers. Compared with the early record of Blue Cross—a remarkable story of progress in itself—our growth has been steady and healthy, and a certain amount of optimism seems justified.

Despite its successful progress, Blue Shield still has far to go to meet its basic obligation—an adequate and effective means of financing physicians' services for as many persons in the community as possible, at a price they can afford to pay.

Let us analyze this objective. Is the protection adequate? It seems as nearly so as we can make it right now, although in time we will probably wish to broaden it. Last year we paid benefits to about one in every ten persons enrolled. Of these cases, under our own Plan (excluding the special surgical program for Bethlehem Steel employees) 60% of the cases involved surgery, 31% were medical, and the remaining 9%, obstetrical. These figures show the importance of our medical coverage, which is not always available elsewhere under Blue Shield, or commercial insurance programs.

Is the price one the great majority can afford to pay? Apparently it is, judging from our own experience and that of Blue Shield Plans in other areas. Whether it could be increased in order to broaden benefits is a question that must some day be studied.

But what of our objective to make Blue Shield available to as many persons as possible? Here we have only scratched the surface of our potential market. We have enrolled only 22% of the Blue Cross membership. A tremendous selling job remains to be done, and this is not easy, since we find that in the majority of large groups we have to replace an existing commercial coverage taken out in the years before Blue Shield was in existence.

One of the most important features of Blue Shield —one that could and should be the most important —is the service benefit feature whereunder participating physicians agree to make no additional charge for covered services to individuals and families with annual incomes less than the pre-established amounts.

This is the one factor that distinguishes Blue Shield from commercial health insurance.

To persons in the lower income brackets, this service benefit feature means full protection against unexpected medical bills. They get the best medical care, and are able to budget for it in advance through membership in Blue Shield.

In agreeing to provide services at pre-determined fees to persons in the lower income brackets, the physician is not deviating from the established practice of charging patients according to their ability to pay. At the same time he is providing real service to the community.

The phenomenal growth of Blue Shield—24 million members nationally—has been one of the chief factors (along with the growth of Blue Cross) in persuading the proponents of Federal compulsory health insurance that the provision and financing of medical care for the self-supporting population can be achieved on a voluntary basis.

Blue Shield is our creation. It is controlled here and elsewhere by the medical profession. We collect the money and undertake to provide the services of qualified physicians, which is right and proper since our interest is paramount. But with our control goes responsibility, and with responsibility goes opportunity. If any other agency, in or out of Government, undertook to provide the public with professional services, we would lose control of medical practice.

[1] Read before the House of Delegates of the Medical and Chirurgical Faculty, April 27, 1953.

The continued success of Blue Shield will depend entirely upon the degree of our voluntary cooperation and participation. Hence, we must endorse Blue Shield to our patients and friends. By so doing we will bring the program closer to its objective—an adequate and effective means of financing medical care for as many persons as possible at a price they can afford to pay—and we will have rendered a real service to our patients, to the community, and to ourselves.

AMA OFFERS TWO NEW EXHIBITS

AMA NEWS NOTES, VOL. 2, NO. 5

Two new exhibits will be available for showing at state medical society meetings this fall, the Bureau of Exhibits reports. One exhibit—"Accidental Poisoning in Children"—is based on authentic fatal accident cases, while the other—"A Medical Service Program for your Community"—covers various activities of medical societies and voluntary and public health agencies.

The exhibit on accidental poisonings, from the Committee on Pesticides, emphasizes points of danger in the home, yard and selected farm locations. When staffed with trained personnel, this makes an effective showing at public gatherings, state and county fairs, food and home shows. Subjects included in the exhibit developed by the Council on Medical Service range from care of the chronically ill to hospital construction, physician placement, and rehabilitation.

Both exhibits require space 10 feet long and six feet deep—or, if shown in extended fashion, 20 feet long and three feet deep. Requests for bookings may be made to the Bureau of Exhibits after June 15.

HOW TO GET A DOCTOR—BOOKLET AVAILABLE SOON

AMA NEWS NOTES, VOL. 2, NO. 5

A new pamphlet—"A Doctor for your Community"—will be published some time in June by the American Medical Association. This booklet, directed toward communities seeking a physician, describes briefly the problems involved in obtaining a doctor, the things a community can do to attract and keep a doctor, and examples of what has been done elsewhere. A joint project of the Council on Medical Service, Council on Rural Health and Department of Public Relations, the booklet will be available to state medical societies for distribution to communities listed with their placement services.

In addition, the Council on Medical Service has compiled information from numerous state placement services in a reprint which will be especially useful to state societies interested in expanding their activities in this field.

Woman's Auxiliary to the Medical and Chirurgical Faculty

MRS. CHARLES H. WILLIAMS, *Auxiliary Editor*

ANNUAL REPORTS OF THE COMPONENT AUXILIARIES

WOMAN'S AUXILIARY TO THE BALTIMORE COUNTY MEDICAL ASSOCIATION

MRS. THOMAS E. WHEELER*

Our Auxiliary has been very active since the last meeting of the Woman's Auxiliary to the Medical and Chirurgical Faculty, with the following accomplishments to report:

1. In June of 1952, the Auxiliary was invited to attend a joint meeting with the Baltimore County Medical Association at which time our second student nurse received her scholarship award; and awards were presented for the Medical Association Emblem. (A contest was sponsored in the Baltimore County High Schools by the Baltimore County Medical Association.)
2. September found our Auxiliary very busy with an exhibit at the Timonium Fair. Free movies, "Your Doctor," and "Frontier in Medical Research," were shown. Two student nurses from the University Hospital were on duty to give free blood pressure examinations. Approximately 10,000 pieces of literature were distributed by two doctors' wives, on a four hour shift. Baltimore City Auxiliary members joined with the Baltimore County Auxiliary to make this project a success.
3. The Auxiliary again joined the Baltimore County Medical Association as luncheon guests of the Rosewood State Hospital. Election of officers was then held.
4. Executive meetings were inaugurated to be held as necessary to expedite business so as to allow the regular meetings of our Auxiliary free for more social programs.
5. March 28, 1953, was set aside for our third annual Doctors' Day celebration. A dinner-dance was held at the Sheraton-Belvedere Hotel. The evening proved to be most enjoyable, with a wonderful attendance and a great financial success. Proceeds of this affair will be used to sponsor our third nurses' scholarship fund.
6. Our Nurse Recruitment Chairman is very active supplying Future Nurses of America Clubs; in the Baltimore County High Schools; with program material and suggestions, also in starting clubs in any schools where students are interested in nursing.

* President.

WOMAN'S AUXILIARY TO THE BALTIMORE CITY MEDICAL SOCIETY

MRS. ALBERT E. GOLDSTEIN*

The Woman's Auxiliary to the Baltimore City Medical Society has had a very interesting and constructive program for 1952 and 1953.

The Nurse Recruitment committee has been very active throughout the year.

A two part program has been planned:

1. To interest young girls in nursing as a career.
2. To make it possible for inactive registered nurses to offer their services to the community.

A film is being made on nurse recruitment that will be of interest to high school girls. The medical and nursing directors of all Baltimore hospitals with training schools were contacted and the State Nurses Association was represented. Mr. Carroll D. Hill, Director of Union Memorial Hospital accepted the chairmanship of finance. Twenty-five hundred

* President—April, 1952–1953.

dollars was contributed which will cover the cost of the film.

Four general meetings were held during the year when interesting speakers talked.

The Civil Defense Committee has been engaged in an active program.

The Ways and Means Committee arranged a fashion show and card party in November.

The Medical Research Committee gave support to Baltimore County Auxiliary at Timonium Fair.

The committee on Legislation worked with the Ballot Battalion on Election day, aiding mothers to vote.

Hospitality Committee was busy throughout the year serving lunch at our general meetings and serving coffee and doughnuts to the Doctors at Baltimore City Medical Society meetings.

The final climax and high light of our year was the Medical Chirurgical Ball on the eve of the Annual Faculty Meeting, April 27, 1953.

WOMAN'S AUXILIARY TO THE FREDERICK COUNTY MEDICAL SOCIETY

MRS. P. S. LANSDALE*

The Woman's Auxiliary to the Frederick County Medical Society has held three meetings of the scheduled four for the fiscal year 1952–1953 (October through May) and to date has the following record:

Total number of members, twenty-six. This includes three new members.

Mrs. Forbes Burgess, Chairman of "Today's Health" reported a total of fifteen subscriptions since October, and Mrs. Russell Guest, Chairman of the National Bulletin, reported a total of eleven subscriptions this year.

Our main source of funds was the Fashion Show-Luncheon at the Francis Scott Key Hotel on December 10th from which we derived $235.00 profit. It was our privilege at this time to have as our guest our State President, Mrs. Charles H. Williams.

The expenditures include the purchase of a 21 inch Admiral television set (table model) for $255.00 which was presented to the Nurses' Home of the Frederick Memorial Hospital for their recreation room, a repair bill of $20.35 for the movie projector which is used in the classroom to further the education of the nurses, along with new films to be shown.

The Auxiliary also voted to increase the amount of the Society Award, given each year at graduation, from $10.00 to $25.00, to the student nurse having the highest theoretical average and record of distinguished all-round efficiency in nursing.

Mrs. Byron D. White, Chairman of Nurse Recruitment, reported that Miss Clements, Director of Nurses at Frederick Memorial Hospital, has a total number of fifteen signed and accepted applications for the new training class in September and that she hopes to have twenty-five by that time. Mrs. Clements and Mrs. White, together with Mrs. Frank Worthington who is also on the Nurse Recruitment Committee, have visited High schools throughout the county talking on the subject and showing the students through the Hospital and Nurses' Home and inviting them to teas.

* President.

WOMAN'S AUXILIARY TO THE MONTGOMERY COUNTY MEDICAL SOCIETY

MRS. J. M. BANKHEAD*

1. Regular Luncheon meetings held on first Tuesday of each month September through April, three members serving as hostesses.

2. Programs, each Committee Chairman had charge of one program, at which time she presented her project.

3. Membership; 35 members for this year, an increase of seven over the previous year—an average attendance of fifteen to eighteen.

4. Auxiliary members sponsored a "Get Acquainted" dance for the Doctors on the third Tuesday in October, with one hundred couples in attendance.

5. Name badges have been typed to identify the members of the County Society and a posting board is used to hold them when not in use.

6. Diabetic Detection Clinic—two Auxiliary members served on this Committee.

7. Nurse Recruitment Committee now in process of choosing a candidate for our nursing scholarship.

8. Question Box exhibit held at Montgomery County Hospital Supper in August 1952.

* President.

9. Doctors' Day observed by Auxiliary, members delivering two hundred red carnations to the Doctors in Montgomery County.

10. Today's Health—seven subscriptions.

WOMAN'S AUXILIARY TO THE PRINCE GEORGE'S COUNTY MEDICAL SOCIETY

MRS. DAVID S. CLAYMAN*

This past year, the Auxiliary to the Prince George's County Medical Society, has endeavored to formulate a program to cover as many phases of Auxiliary work as possible. I am pleased to present the following summary of the year's work:

In September 1952 the Auxiliary celebrated its TENTH ANNIVERSARY with a luncheon held at the Prince George's Golf & Country Club. The guest speaker was Mrs. Charles Williams, our State President.

On October 1952, the County Fair was held at Upper Marlboro, the County Seat. At the Fair, the Auxiliary showed movies on NURSE RECRUITMENT, HOW TO CATCH A COLD, SEE YOUR DOCTOR EARLY, and several other films which were obtained from the American Medical Association in Chicago. These films were later shown in the Upper Marlboro High School with special emphasis on the movie for NURSE RECRUITMENT. The Auxiliary also had two large displays set up at the Fair, one on MATERNAL AND CHILD CARE, and the other on DANGERS OF SELF DIAGNOSIS AND SELF MEDICATION. These displays were manned at all times by members who distributed leaflets on subjects pertaining to HEALTH and to the various medical bills which were under discussion at the time.

Last year the Auxiliary gave a worthy girl in the County a scholarship to a school of nursing of her choice. This girl was selected on the basis of her scholastic ability and aptitude by examinations given by the SCHOOL BOARD. The Auxiliary voted to do the same thing this year. A THEATRE PARTY was held and a profit of $460.00 was made, out of which $300.00 will be used to take care of the scholarship.

In March of this year, the Medical Society and the Staff of Prince George's General Hospital were surprised to learn that an enterprising young delegate, a member of the County Delegation to the General Assembly, had written and was trying to put over a bill in Annapolis which would have placed the administration of the Hospital under direct political control. When the Medical Society, Hospital Guild and other interested groups heard of this bill, it was decided to protest against it. The Auxiliary sent letters to each Delegate and to the Senator of the County protesting the proposed bill. In addition, I went to Annapolis with the representatives of the Medical Society to attend the hearing upon the proposed bill. Upon hearing some of the delegates laughingly sneer and define the Auxiliary as "THE DOCTORS BOSSES AT HOME," I decided to speak up. I stated who made up the membership of the Auxiliary, what the Auxiliary had done for the hospital in the past, what it was doing for it at present and what it hoped to do for it in the future, then ended by voicing the protest of fifty active members against the bill. I would like to think that this helped even a little to defeat the proposed bill.

Phone calls were made to the members of the county delegation and to our Senator when we received word that the Bill on DOCTORS' DAY was due to come up for consideration. When we were notified that the bill was coming up definitely on March 30th, the Auxiliary sent telegrams to these same men urging the passage of the Measure.

On DOCTORS' DAY, The Prince George's General Hospital furnished a beautiful buffet luncheon for the doctors. The Auxiliary sent out the invitations, set up the tables, helped serve the luncheon and presented each doctor as he entered the hospital with a beautiful red carnation. In addition, I was interviewed by Mr. Ernie Tannen, the Program Director of Radio Station WGAY, Silver Spring, Md., on the subject of DOCTORS' DAY.

All of these endeavours received good notices from the press.

In addition we have had various programs throughout the year, such as a Musicale, a Book Review, afternoon of cards, etc. At Christmas time, the Auxiliary supplied groceries and clothing to a needy, local family. The Auxiliary paid for $100.00 worth of magazines and periodicals for the hospital. We subscribed to the BULLETIN and we took sub-

* President, Selma H. Clayman (Mrs. David S. Clayman).

scriptions to TODAY'S HEALTH, sending some to the schools.

My sincere thanks to the Officers and the Committee Chairman for a most cooperative year, and to the individual members for their staunch, wholehearted support thereby contributing to an enjoyable and a successful year.

ANNUAL REPORT OF THE WOMAN'S AUXILIARY TO THE WASHINGTON COUNTY MEDICAL SOCIETY

MRS. GERALD W. LEVAN*

During the past year the Woman's Auxiliary to the Washington County Medical Society consisted of an active membership of fifty-eight out of an eligible sixty-five doctors' wives. Within the past months we gained five new members and our total membership today is sixty-three.

Nurse Recruitment is our one big project and we have awarded one (1) full scholarship in nursing and are prepared to grant partial scholarships to two girls (twin sisters) in the near future when they are accepted by the Nursing School. On March 24th, we will have a Tea for 200 high school girls, following their conducted tour of the Hospital and Nurses' Home, in our effort to increase their interest in nursing careers. Posters and pamphlets on nursing have been distributed in the offices of professional men, in the schools of the county, and in lunch rooms, Youth Centers, and wherever the students congregate. To add to our Nursing Scholarship Fund, we are holding an All-Day Rummage Sale on March 27th, and with each and every Auxiliary member contributing to this, we expect to make a rewarding addition to our treasury.

In September 1952, the Auxiliary manned a Health Booth during the week of the County Fair, at which time medical and health pamphlets were distributed and scientific exhibits shown. Our members assist in all health drives, such as Cancer, Polio, Red Cross, etc. Last fall, we sent more than 200 lbs. of medical supplies and literature to New York for overseas shipment.

At present we have 38 National Bulletin subscribers and 19 subscriptions to "Today's Health," and hope to increase these figures by the end of this year.

A chairman was appointed in January to work for the A.M.E.F.

With March 30th drawing near, we are deep in our plans for the annual observance of Doctors' Day when we will honor our doctors of Washington County.

* President.

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FACULTY OFFICE AND LIBRARY HOURS

August—9 a.m. to 4 p.m.

Ancillary News

PHARMACY SECTION

MARYLAND PHARMACEUTICAL ASSOCIATION

L. M. KANTNER, PHAR. D.[1]

Journal Representative

A story is told, the veracity of which is not vouched for, but whether true or not, it does reveal the thinking of some people—likely a good proportion of the public relative to the procurement and use of a class of drugs that have been developed exclusively for use under medical supervision.

The story relates to Mrs. Blank who took her young child to a physician, telling him she wanted a "shot of penicillin" given the child for a cold. The physician's reply to the request was: "When did you begin directing your physician what medication to use? I will give the child an examination and if *I* consider penicillin is the proper medication, I will administer it. Otherwise, my judgment will dictate what treatment your child requires."

If this physician had the disposition alike to some people I know, under ordinary conditions, the child would not have gotten penicillin medication for the reason the physician would not have enriched Mrs. Blank's ego by allowing her to believe that after all "he did what I told him."

This story is related because of the number of people who have acquired a superficial knowledge of modern drugs through dramatic magazine articles, press releases written by enthusiastic writers, etc., and are quite willing to make guinea pigs of themselves.

Pharmacists report that many people make requests for drug products that can be dispensed only on prescriptions. Frequently, the request is amplified by "my doctor told me to get it." In many cases, the customer is untruthful by involving the physician; while at other times, they are truthfully carrying out a physician's instructions.

It would be ridiculous to claim all pharmacists are ethical in their professional or commercial conduct—they are as human as those in any vocation. There is a fringe who, regardless of consequences, will ignore any and all laws of decency and ethics. On the other hand, all are subject at times to being off guard, and a drug is supplied upon such requests as above mentioned by those whose professional conduct is unquestioned. However, under the law there is no immunity for the sale of what are termed dangerous drugs without a prescription.

The sale of such drugs as the barbiturates, amphetamine, sulfa drugs and thyroid by the unscrupulous often are not detected unless serious consequences occur from their unsupervised use. Often those who have suffered ill effects from the use of these drugs, particularly the barbiturates, will not divulge where they procured them because they evidently want to protect their suppliers, and to continue in good graces for future supplies.

For no other reason than for the public welfare have these drugs, by legislation, been taken out of reach except by physician's administration. No laws are entirely effective, nor do they successfully serve the purpose; but it is rational reasoning if there were not controls over the distribution of dangerous drugs, tragedies from their use would be comparable to their cures.

Reports are received that more and more physicians are including on their prescriptions authorization to refill a definite number of times, or signifying the prescription cannot be refilled. Adopting this policy obviates much annoyance to the physician, pharmacist, as well as the patient, when the latter desires a prescription refilled that requires the prescriber's authorization.

[1] Secretary, Maryland Board of Pharmacy.

POSTGRADUATE LECTURE COURSE IN BASIC SCIENCES TO BE OFFERED AGAIN IN 1953-54

DIETRICH C. SMITH, PH.D.*

For the third consecutive year, 1953–54, a course in THE BASIC SCIENCES AS THEY APPLY TO THE PRACTICE OF MEDICINE will be offered, according to an announcement by the Postgraduate Committee of the University of Maryland School of Medicine. The decision to repeat the course next fall was based on the response to a questionnaire sent to all those enrolled in 1952–53. When asked whether, in their opinions, the course should be repeated, all those who replied, unanimously recommended such action.

A considerable body of experience has now accumulated which will be valuable in formulating plans for the coming year. The questionnaires contained many helpful suggestions, all of which have been given serious consideration by the Committee and the lecturers responsible for the course. Special attention, however, is called to the fact that the course is concerned primarily with the fundamental biological concepts on which the practice of medicine rests and how these concepts apply to such practice. It is not intended to give specific instructions as to how to proceed in definite clinical situations. Such instruction is available at the numerous clinical pathological conferences, seminars, symposia, etc., sponsored by local hospitals and medical societies.

Judging from past experience, the course appeals to two groups of physicians; younger men who take it as part of the residency training program offered by their hospitals, and general practitioners who want to be brought up to date in the ideas that are germinating in the basic sciences. Particular stress will be laid on the changes which have occurred in this area within the last ten years or so. In this way, the course becomes a helpful review in the basic sciences for those men who are preparing for their board examinations.

Biochemistry, physiology, pharmacology, embryology, immunology, neuroanatomy, neurophysiology and histology are included, while gross anatomy and pathology are not. The latter two subjects are thoroughly covered in other courses in these special fields and offered by the University of Maryland School of Medicine. In the past, lecture schedules have included such subjects as the physiology of hypertension, its pharmacological and surgical control, natural and immune mechanisms of host resistance, practical aspects of neurophysiology and the pharmacology of the antibiotics and antihistaminics, to mention but a few.

Thirty-two two-hour meetings will be held, as in the past, on Wednesday at 4:00 P.M. except during University holidays, on the 2nd floor, Bressler Building, 29 S. Greene Street. The greater part of the enrollment usually consists of physicians in training in the Baltimore area, but any qualified and interested physician is welcome. Many local practitioners have taken advantage of this opportunity, and the course is open to the graduates of any approved medical school on application to the Committee.

The lecture schedule as well as the lecturers for the coming session will be announced shortly and may be obtained on request from the Office of the Postgraduate Committee, 6th floor Bressler Building, 29 S. Greene Street, Baltimore 1, Maryland. A detailed prospectus, further information and application blanks may be obtained from the same source. Telephone inquiries are also welcome. Call Mrs. Elizabeth Carroll, Plaza 2-1100, Extension 278. Tuition is $50.00 and is payable in advance. Registration will be held on September 21 and 22, 1953 from 9:00 A.M. to 4:00 P.M. at the Postgraduate Office, and the first lecture will be held on Wednesday, September 23, 1953.

Those who attend at least 27 of the regularly scheduled meetings shall be eligible, on vote of the Postgraduate Committee, to receive a certificate. Such certificates are issued on request to those regularly enrolled, on payment of an additional fee of $7.50 and on signing a statement that the required number of lectures have been attended.

* Vice-Chairman and Assistant Director, University of Maryland, School of Medicine Postgraduate Committee.